JOURNAL OF

The American Geriatrics Society

Volume II *June 1954* Number 6

CONTENTS

Obituary. Willard O. Thompson 332
Chronic Cor Pulmonale and Its Management. *M. S. Segal, S. S. Radovsky and A. Salomon.* 335
The Management of Edema in the Elderly. *G. E. Miller* 344
Complications of Diabetes in Older Patients. *J. I. Goodman* 351
Methonium Compounds in the Treatment of Hypertension. *J. B. Wolffe, M. B. Walkow, J. H. Nagler and J. Anastasi* 359
Medical Aspects of Physical Treatment in Geriatrics. *H. Dinken* 367
Patterns in Aging. *J. T. Freeman* 371
Some Aspects of the Gastric Cancer Problem. *S. F. Marshall and K. W. Warren* 377
Diagnostic and Etiologic Considerations in Deafness in Older Persons. *L. W. Alexander.* 386
Problems Arising in the Management of Senile Cataract. *E. I. Lipsius* 396
Management of Benign Hypertrophy of the Prostate. *R. H. Flocks* 400
International Association of Gerontology. Provisional Program of Third Congress 405
Report of Research Committee on Biology and Medicine 410
American Geriatrics Society—Program of the 1954 Annual Meeting 411
Gerontological Society, Incorporated.
The 1954 Annual Meeting 415
Winners of 1953 Schering Award for Medical Students 415
Book Review 416

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JOURNAL OF

The American Geriatrics Society

Official Journal of the American Geriatrics Society

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Subscriptions. The *Journal* is published monthly; twelve issues (Jan.–Dec.) constitute a volume. Subscriptions are sold on a volume basis, prorated if back volumes are not available. The subscription price is $10.00 per volume inside the Postal Union. Foreign postage is 75 cents extra. Subscriptions for members of the American Geriatrics Society are included in the annual dues which are $15.00. Single copies, when available, are $1.25 per copy. Publisher must be notified 60 days in advance of change of address. Undeliverable journals will be destroyed.

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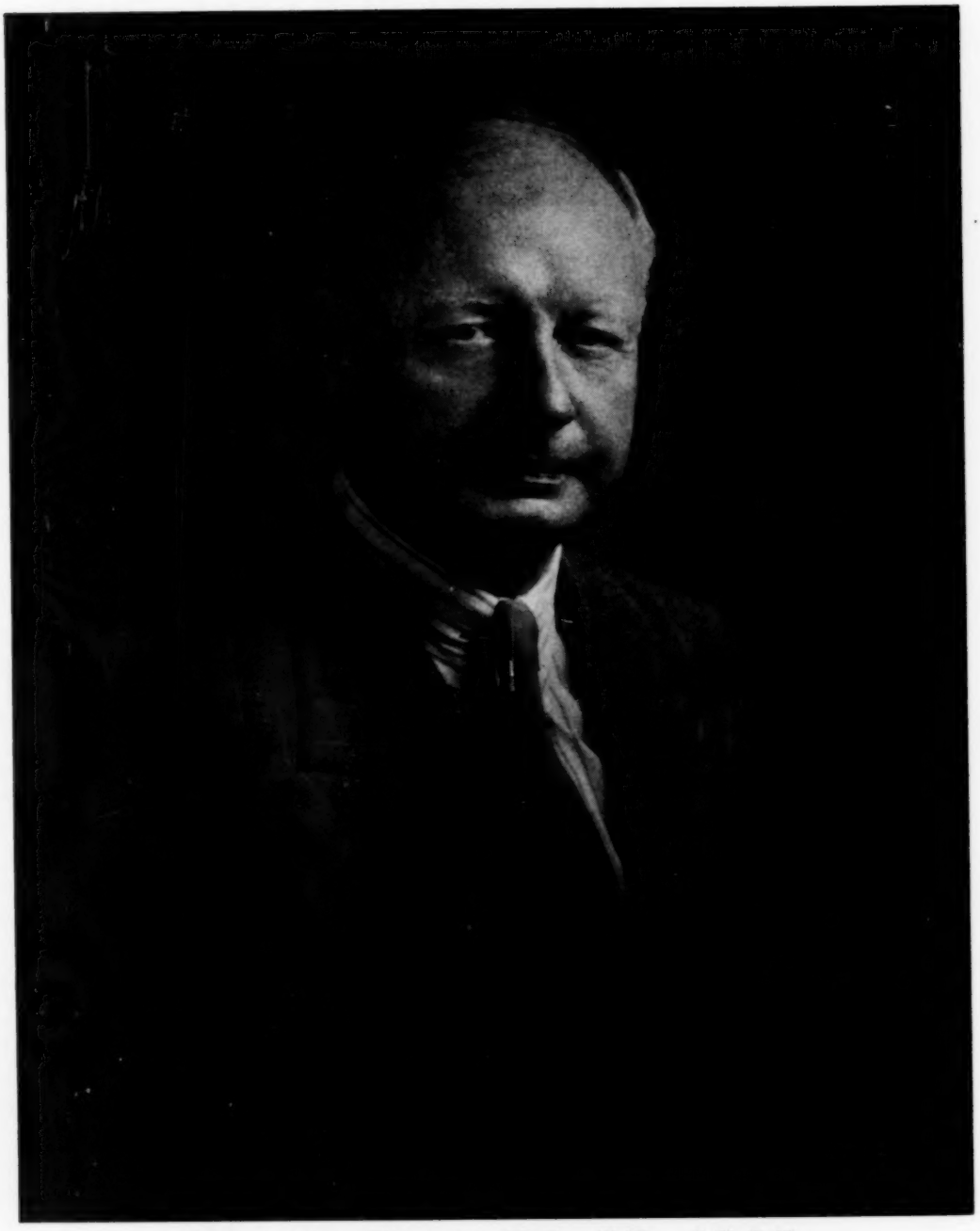

Willard Owen Thompson, B.A., M.D., F.A.C.P.
1899—1954

Willard Owen Thompson, B.A., M.D., F.A.C.P.

February 17, 1899–March 23, 1954
President, American Geriatics Society
Editor, Journal of the American Geriatrics Society

Doctor Thompson, well beloved President of our Society and Editor of this Journal died at Henrotin Hospital, Chicago, on March 23, 1954.

One of the most distinguished men in American medicine, he was known to his many friends as Willard. A dynamo of energy, he had never learned to refuse an appeal for help with any problem having to do with the betterment of the medical sciences.

He was an indefatigable worker for the American Geriatrics Society and our Journal. It was largely through his influence that our Society has gained its prominent place in medicine today and it was almost entirely through his energy and hard work that this Journal was founded last year.

His untimely death surely may be ascribed to the fact that Willard was tireless in his work for our Society, for this Journal, for the University where he served as Clinical Professor of Medicine, and above all, for his many patients. He was constantly doing good things for other people with little thought of himself.

Although he had reached distinguished heights in the practice of medicine where many would have justifiably restricted their efforts to seeing patients only in their office, right to the end Willard insisted upon responding to calls from his patients at any hour of the day or night. The last time I saw him, only a few weeks ago, at about nine o'clock in the morning, he had just returned home after having been up all night with one of his desperately ill patients. While there is nothing very unusual about this in the practice of medicine, in Willard's case it was a tragic sacrifice to his ideals since he knew that such strains upon his failing resources courted disaster. He had known for some time that he was suffering from hypertension and related cardiovascular disease but refused to forsake the many causes he espoused. Truly it may be said that he died as a result of his insistence on continuing to carry the heavy burdens he had imposed upon himself.

He was always willing and glad to serve his profession by every means at his command. All who knew him will testify that whatever he undertook to do he did well.

He was a fine doctor, beloved by all of his patients. He was a fine teacher, always to be remembered by the many hundreds of students who have come under his stimulating influence during the past two and a half decades at the University of Illinois School of Medicine.

Willard was devoted to his family and it was quite usual at medical meetings for him to bring his entire family, his wife, three sons, and one daughter, in order that they would remain close together.

He was a warm and true friend and will be sorely missed by those who had come to know him best and to love and respect him as a man representing the highest ideals in his chosen profession.

While no one can ever take his place in our affections, we realize that the work so ably initiated by him for our Society and our Journal must be continued. We, upon whose shoulders some of these burdens have fallen, dedicate our energies to maintain those high standards set by our dear friend and colleague Willard Owen Thompson.

EDWARD HENDERSON, M.D.

To those of his many friends who would like more complete information concerning the many activities of this distinguished man of medicine we list below his curriculum vitae as it appears in the current *Who's Who in America*:

Thompson, Willard Owen, physician; born Fredericton, New Brunswick, Canada, February 17, 1899; son of Samuel Stirling and Mary (Owen) T.; B.A., Dalhousie University, Halifax, Nova Scotia, 1919; M.D., Harvard, 1923; married Phebe K. Christianson, June 21, 1923; children—Willard Owen, Frederic Christianson, Nancy Kirsten, Donald McRae. Came to United States 1920, naturalized, 1937. Engaged in practice of medicine since 1925; interne Boston (Massachusetts) City Hospital, 1923–25; research fellow in medicine Harvard Medical School, 1925–28, graduate assistant in medicine Massachusetts General Hospital, 1925, research fellow in medicine, 1926–29, in charge of metabolism laboratory, 1926–29; assistant in chemistry, Harvard, 1927; assistant clinical professor of medicine, Rush Medical College, University of Chicago, 1929–35, associate clinical professor 1935–41; associate professor of medicine, University of Illinois College of Medicine, 1941–43, professor of medicine, 1943–45, clinical professor of medicine since 1945; research associate in pathology, Cook County Hospital, 1934–40; associate attending physician, Presbyterian Hospital, Chicago, 1930–46; attending physician, Grant Hospital, Chicago, since 1947; attending physician, senior staff, Henrotin Hospital, Chicago, since 1947; attending physician, University of Illinois Research and Educational Hospitals, since 1945. Member Chicago Association of Commerce and Industry, Certified by American Board of Internal Medicine, 1937; diplomate, National Board of Medical Examiners, 1925. Fellow American College of Physicians and American Medical Association, American Geriatrics Society (president), Society for Research in Child Development; member Association of American Physicians, American Society for Clinical Investigation, Central Society for Clinical Research, Endocrine Society (vice president), Society for Experimental Biology and Medicine, American Goiter Association (president), American Association for the Advancement of Science, American Association of the History of Medicine, American Diabetes Association, American Society for Research in Psychosomatic Problems, American Therapeutic Society (vice president), Gerontological Society, American Society for the Study of Sterility, American Heart Association, World Medical Association, American Medical Writers' Association, American Genetic Association, American Academy of Compensation Medicine, Chicago Medical Service (board of trustees), Chicago Association of Commerce and Industry (executive forum), Institute of Medicine of Chicago, Chicago Society of Internal Medicine, Society of Medical History of Chicago, Chicago Medical Society (president), Associated Diplomates of the National Board of Medical Examiners (president), Mississippi Valley Medical Society (recipient distinguished Service award, 1952; president, trustee, executive committee), Illinois State Academy of Science, Illinois State Medical Society, Sigma Xi, Pi Kappa Epsilon. Republican. Presbyterian. Clubs: University, Racquet, Harvard (vice president for Chicago). Contributor, numerous articles to medical journals and textbooks. Managing editor Journal of Clinical Endocrinology and Metabolism; editor Journal of American Geriatrics Society, American Lectures in Endocrinology; associate editor American Practitioner; editorial board Mississippi Valley Medical Journal; adv. editorial board Journal American Academy of General Practice (GP). Home: 1430 Lake Shore Drive, Chicago 10. Office: 700 North Michigan Avenue, Chicago 11.

JOURNAL OF

The American Geriatrics Society

Official Journal of the American Geriatrics Society

VOLUME II *June, 1954* NUMBER 6

CHRONIC COR PULMONALE AND ITS MANAGEMENT

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By definition, chronic cor pulmonale is not a primary condition; it is heart failure secondary to disease of the lungs or their blood vessels. The primary pulmonary disease must be such that it interferes with the passage of blood through the pulmonary circuit, thereby putting an undue strain upon the right chambers of the heart. Interference with blood flow can be anywhere between the pulmonic valve and the left atrium but it usually originates in either the pulmonary arterial tree or capillary bed, primary obstruction of venous flow being unknown. Although chronic cor pulmonale has been invariably bound to pure right ventricular hypertrophy, it has recently been shown that in many instances the left ventricle and the systemic circulation are likewise involved.

ETIOLOGY

Chronic conditions which affect principally the pulmonary arteries include thrombosis and recurrent embolism, sickle-cell anemia, schistosomiasis, and pulmonary arteritis (including collagen disease and thromboangiitis obliterans) (1). Conditions which affect chiefly the pulmonary capillaries include chronic obstructive emphysema, and pulmonary fibrosis or granulomatosis from a variety of causes, including sarcoid, pneumoconiosis, scleroderma, adenomatosis and beryllium poisoning. Secondary arterial changes are seen frequently in this latter group.

A third group are those conditions which restrict pulmonary expansion and ventilation, and which produce interference with pulmonary blood flow and thus cor pulmonale. They include severe thoracic-cage deformities resulting from infantile paralysis, spinal tuberculosis, congenital or developmental anomalies such as funnel chest, post-thoracoplasty procedures, or extreme pleural thickening from empyema or hemothorax. In these conditions, some degree of emphy-

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TABLE 1

*Causes of Chronic Cor Pulmonale**

A. Primarily arterial
   1. Recurrent pulmonary embolism
   2. Thrombosis of pulmonary artery
   3. Sickle-cell anemia
   4. Schistosomiasis
   5. Arteritis—thromboangitis obliterans; disseminated lupus; rheumatic arteritis; polyarteritis nodosa; Raynaud's disease; Ayerza's disease
   6. Idiopathic
B. Primarily capillary and parenchymal
   1. Chronic obstructive emphysema
   2. Sarcoidosis
   3. Pneumoconiosis; beryllosis
   4. Tuberculosis
   5. Scleroderma; interstitial fibrosis
   6. Diffuse adenomatosis
C. Compressive
   1. Thoracic cage deformities—kyphoscoliosis; funnel chest
   2. Thoracoplasty
   3. Pachypleuritis—hemothorax; empyema

* Modified from Segal, M. S., and Dulfano, M. J.: Chronic Pulmonary Emphysema. Physiopathology and Treatment. New York, Grune & Stratton, 1953.

sema is frequently present, but additional factors probably are involved. Kinking of the major blood vessels and compression of the pulmonary parenchyma have been suggested as causes, but neither has been satisfactorily shown to be capable of raising pulmonary arterial pressure experimentally.

Idiopathic pulmonary hypertension has also been described as a primary cause of chronic cor pulmonale, and it should be included among etiologic factors.

PATHO-PHYSIOLOGY

Chronic pulmonary emphysema is the most common cause of chronic cor pulmonale (2). Apparently, as alveolar walls rupture and disappear and many of the capillaries within them are lost as channels for blood flow from pulmonary artery to pulmonary veins, there is a rise in pressure in the pulmonary artery in what may be regarded as an effort of the body to maintain blood flow through a narrowed passageway. If this increase in pressure persists, organic narrowing of the arterioles may occur (as in the development of arteriosclerosis in systemic hypertension) and add to the burden of the right heart. A further insult is the occurrence of hypoxia, the result of the disappearance of the pulmonary parenchyma and consequent diminution of gas exchange (3). This produces a rise in cardiac output and a further increase in pulmonary hypertension. At a critical point, when the pulmonary arterial resistance is three times the normal or higher, hypoxia causes an actual fall in cardiac output (4). At this stage, when the overburdened myocardium cannot deliver the required blood, cardiac failure

of serious significance often develops. A second consequence of hypoxia is the development of polycythemia, which is compensatory in name only, and which, by increasing blood viscosity, adds further to the distress of the myocardium. Other diseases causing chronic cor pulmonale behave in a similar manner—pulmonary hypertension and hypoxia combining to bankrupt the right ventricle.

CLINICAL MANIFESTATIONS

The clinical picture of chronic cor pulmonale varies according to the underlying etiologic condition. Unfortunately, the early diagnosis of chronic cor pulmonale can be confirmed only by finding (through cardiac catheterization) an elevated pressure in the pulmonary artery. The most important aid to making a diagnosis is to suspect the presence of the condition when congestive failure develops in a patient with severe emphysema, or with one of the other pulmonovascular diseases listed previously. However, certain features are common to most patients with full blown chronic cor pulmonale. Most patients have a history of exertional dyspnea but are able to lie flat comfortably; the development of orthopnea may be noted only after many months or even years. They usually have a secondary polycythemia (unless there is chronic infection) which causes venous distention and gives a dusky flush to the head, neck and distal portions of the extremities. The neck veins are usually distended. Examination of the chest reveals limited motion of the rib cage, a low relatively fixed position of the diaphragm, distant breath sounds with sibilant rhonchi and, on occasion, moist rales. The chest may be increased in its anterio-posterior diameter and the rib spaces widened. Tachypnea, tachycardia and a slight elevation in blood pressure are almost invariably present. There is cardiac enlargement, but it may not be clinically apparent because of emphysema. A systolic heave just to the left of the sternum, and an accentuated and sometimes split pulmonic second sound are frequently found. Late in the disease, the liver is usually palpable and tender, and there is commonly edema of the lower extremities. The vital capacity is generally reduced and the time relationships altered, the entire respiratory cycle lasting longer than usual. Occasionally one may note a normal or higher than normal vital capacity, but the time required for performance of the test is prolonged.

Examination of the blood constituents, in addition to showing polycythemia, reveals arterial oxygen unsaturation, and sometimes an elevated carbon-dioxide combining power and a diminished level of chlorides in the venous plasma. X-ray examination of the chest may show the primary pulmonovascular disorder which is the cause of the chronic cor pulmonale. Also, early in the course of the disease one may detect the presence of a prominent pulmonary conus, which will be apparent first in the right anterior oblique position, and later in the ordinary posterior-anterior view. It may be differentiated from that occurring in patients with mitral stenosis, by the presence of an indentation below it, because of the fact that in early cor pulmonale the left atrium is not enlarged (5). Later the characteristic, triangular shaped "cor pulmonale heart" will be seen. Evidence of pulmonary congestion may range from moderate to absent on

the chest film. The electrocardiogram may show the pattern of right ventricular hypertrophy, with a qR of rSr′ pattern in the right precordial leads, and an rS or RS pattern in the left precordial leads. On the other hand, a vertical electrical axis may be the only unusual feature of the QRS pattern. The P waves may be unusually high and peaked, and the T waves in V_2 and V_3 may be inverted. Determinations of venous pressure and circulation time may be helpful; the former will be elevated, and the latter will usually be normal or only slightly prolonged.

Occasionally, because of exhaustion of the patient's respiratory musculature and loss of sensitivity of the respiratory center to carbon dioxide stimulation (centrogenic drive), the normal increase in ventilation in response to a rise in the arterial blood carbon-dioxide content and tension, will not occur (6, 7). The patient's subsequent respiratory efforts will then depend largely upon stimulation of the chemoreflex centers (carotid and aortic bodies) due to the deficiency of oxygen (hypoxia). If this stimulus to respiration is disturbed by inadvertent correction of the hypoxia, oversedation or severe respiratory infection, respiratory acidosis will then develop, with a measurable drop in the arterial blood pH and a concomitant rise in arterial CO_2 content and tension (7, 8, 9, 10). The respiratory efforts will become less and less adequate (hypoventilation), and if the respiratory acidosis persists untreated, may cease altogether.

Respiratory acidosis may occur and progress spontaneously in the course of chronic cor pulmonale. It may be precipitated or made worse by severe respiratory infection or by the administration of excessive amounts of sedatives, opiates or high concentrations of oxygen (8, 9, 11). At first, such treatment will seem beneficial; dyspnea will lessen under the influence of morphia and, with the addition of oxygen, the cyanosis will be replaced by a healthy pink color. Simultaneously, the patient may become stuporous, however, his pupils may dilate (unless morphia has been given), his pulse become full and bounding and respirations become shallow, grunting and ineffectual (12). In effect, the patient will have been "killed with therapeutic kindness." A major reason for making a correct and early diagnosis of chronic cor pulmonale is to prevent this therapeutic paradox which may indeed be fatal.

TREATMENT

The treatment of chronic cor pulmonale, once it has developed, is similar to that for any type of heart failure. However, attention must be paid to the management of the underlying etiologic disorder, particularly the associated bronchoconstriction and infection. Digitalis, salt restriction and oral or parenteral mercurial compounds in conjunction with ammonium chloride are indicated, more or less in that order, for the heart failure.

The value of digitalis in the treatment of cor pulmonale has been questioned in the past. One reason is that a less dramatic response to digitalis may be obtained in this condition than in other more common types of failure. It is now generally accepted as an important part of therapy, and should be given in full dosage (13). Our policy has been to give a total of 1.2 mg. of digitoxin orally,

in three divided doses at eight-hour intervals, followed by maintenance with digitalis whole leaf in an average dose of 0.1 Gm. per day. An occasional patient will require a larger amount of digitoxin for digitalization, or a larger or smaller amount of whole leaf digitalis for maintenance.

Salt restriction is indicated when there is a tendency toward edema formation, and should be rigid enough to keep the patient free of excess fluid accumulation, but no more than that. Mercurial diuretic agents and ammonium chloride may be used in conjunction with diet, if salt restriction is not sufficient or if edema is already present. The patient's "dry weight" measured daily before breakfast may be used as a guide. A good fluid intake should be maintained during diuresis in order to avoid dehydration in a polycythemic individual, with the danger of consequent thrombosis.

Additional measures that are specific for the underlying pulmonary disease in most patients with chronic cor pulmonale include aminophyllin, aerosol therapy, and phlebotomy. Aminophyllin has a relaxing action upon the pulmonary bronchioles and upon the pulmonary vasculature as well (14). When given in adequate amounts it decreases resistance to air flow into and out of the lungs, clinically diminishes the wheezing, and lessens the respiratory efforts of the patient. Because of this, and also because of the direct effect upon the pulmonary blood vessels, it decreases resistance to blood flow through the lungs, and lessens the work of the right ventricle. A sufficient blood level of aminophyllin is best maintained by administration of the drug intravenously by a continuous slow drip, giving 0.5 Gm. of aminophyllin in a liter of 5 per cent glucose in water every eight hours. Seriously ill patients with a large bronchospastic component should be treated in this manner, therapy being continued until all the wheezing has subsided, which may take several days. Less acutely ill patients may be given aminophyllin rectally, 4 to 7.5 grains in 15 cc. of tap water, every eight hours. The ambulatory patient with chronic cor pulmonale may note increased walking tolerance and less wheezing with an oral aminophyllin preparation such as Cardalin, which permits higher oral dosage with less tendency for nausea.

Aerosol therapy, consisting of the nebulization of a therapeutic solution and its inhalations by the patient, may be found helpful. The therapeutic agent is placed in the Vaponefrin nebulizer, which is connected by rubber tubing to the regulator of an oxygen tank or air pump and nebulized with flow rates of 4 to 6 liters per minute. One half to 1 cc. of a bronchodilator (such as Vaponefrin, 2.25 per cent, or Isuprel, 1:200) in combination with 2 or 3 cc. of a detergent, (such as "Alevaire" (15)), will help to relieve bronchospasm and to facilitate the raising of mucopurulent secretions from the tracheobronchial tree. In patients with large amounts of tenacious sputum, the use of a liquefying enzyme will be of special benefit. We have observed good results with a solution containing 100,000 units of the enzyme, Pancreatic Dornase (16), contained in 3 cc. of diluent. Each aerosol treatment should take about twenty minutes, and may be repeated every three or four hours for as long as necessary.

Phlebotomy is indicated whenever the hematocrit is elevated above 50 per

cent, and should be repeated as often as necessary to maintain a hematocrit of 45 to 50 per cent, drawing off about 250 cc. of blood at a time. Any increase in hematocrit above 50 per cent does not increase appreciably the amount of oxygen carried to the tissues, and adds to the viscosity of the blood and to cardiac work. Following venesection, the pulmonary arterial hypertension may fall to normal levels.

Certain drugs to be discussed may be of value in the treatment of chronic cor pulmonale, but their use may be dangerous and requires caution. These are the sedatives, opiates and high concentrations of oxygen, the dangers of which have already been mentioned in connection with the development of respiratory acidosis (1, 8, 9). Oxygen especially may be valuable, but it is best administered with a flow of 1 liter per minute by nasal catheter at first, increasing the flow by small increments over a period of several days until a rate of 6 liters per minute is reached. When this has been achieved, the ABC[1] plastic oxygen Face Tent may be employed with flow rates of 6 liters per minute, thus ensuring concentrations of oxygen not exceeding 50 per cent. The patient should be inspected frequently at the outset of oxygen therapy for signs of respiratory depression.

There is a great temptation to use opiates to ease the labored breathing of the severely dyspneic patient, and give him a few hours of much needed rest. In general, it is best to avoid the use of morphine. When employed, it should be used only with caution. A single small dose of ⅛ grain may be well tolerated but it should not be repeated. With the opiates and barbiturates, sudden drops in the respiratory rate and depth of respiration should be carefully watched for. The combination of these opiates with the sudden administration of oxygen concentrations above 50 per cent may have very serious results. One of us (M. S. S.) recently observed sudden deaths in 2 patients with chronic cor pulmonale secondary to severe kyphoscoliosis. Both patients had received repeated doses of morphia and mask-oxygen therapy. The deaths followed the typical pattern of respiratory acidosis, hypoventilation, coma and exitus. In the past, such deaths have been attributed to morphine idiosyncrasy. Demerol may be given in a dose not larger than 50 mg., and repeated after an eight-hour interval if indicated, with a somewhat greater range of safety against the danger of respiratory depression.

A mixture of chloral hydrate and sodium bromide, 1.5 Gm. of each, is generally helpful in producing a good night's sleep. If more prolonged sleep is deemed essential, the rectal administration of 20 to 30 cc. of paraldehyde or 60 to 90 cc. of ether, each mixed in an equal amount of olive oil, may prove helpful.

If respiratory acidosis develops during administration of high concentrations of oxygen, the method should be changed to the catheter technique and an oxygen flow of 1 liter per minute. If an opiate has been the cause of the respiratory depression, it should be counteracted at once by the administration of N-allyl-normorphine (Nalline[2]). This drug is pharmacologically related to morphine and

[1] Manufactured by Eliot Medical Plastics, Inc., Lynn, Mass.
[2] Manufactured by Merck & Co., Inc., Rahway, N. J.

has been found in repeated studies (17, 18, 19) to be very effective in counteracting the respiratory depressant effect of the opium derivatives and Demerol. Nalline, in a single dose of 2 to 10 mg., should be given intravenously, followed by the administration of another 5 to 10 mg. mixed with 200–300 cc. of 5 per cent glucose in water by slow intravenous drip.

A further word should be said about the actual treatment of respiratory acidosis, whether spontaneous, or due to the administration of high concentrations of oxygen or opiate. The most important aim in the treatment of respiratory acidosis is to improve pulmonary ventilation, for only by doing so can one rid the blood and tissues of the accumulated excess of carbon dioxide, and permit the respiratory centers of the medulla to regain their normal sensitivity. Any available means of artificial respiration should be used promptly, whether it be an intermittent positive pressure device such as the Bennett or M.S.A. valve apparatus, a Drinker or Emerson type respirator, or even manual resuscitation as an emergency measure.

We have had good results with the use of the Bennett valve[3], which is a device for giving oxygen or compressed air under pressure during the inspiratory phase of respiration (IPPB/I) (1, 20). The flow of gas is initiated by an inspiratory effort on the part of the patient, and proceeds at the pressure selected by the administrator, until an expiratory effort by the patient causes the flow and the pressure to drop immediately to zero. Thus the valve acts as an amplifier for the patient's own inadequate attempts at breathing. The apparatus may also be operated manually by moving a small finger-switch up and down if the patient is apneic. This device should be used with air, or an air-oxygen mixture if available, rather than with oxygen, in the patient with respiratory acidosis. We generally recommend the use of 1 cc. of a bronchodilator aerosol such as Vaponefrin (2.25 per cent) or Isuprel (1:200), with 2 cc. of a detergent such as Alevaire, in conjunction with the intermittent positive-pressure apparatus. These drugs increase the effectiveness of ventilation, and their administration is easily carried out, since a nebulizer is incorporated in the apparatus for this purpose.

Another mechanical aid to respiration is the institution of an emergency pneumoperitoneum (2 to 3 liters of air). This procedure may be used to elevate the diaphragm to a position where more efficient ventilation can be carried out.

A further important aid in the treatment of chronic cor pulmonale is the use of antibiotics. Frequently, there is an element of pulmonary infection which may contribute to the severity of the pulmono-cardiac failure and may even "trigger" the development of respiratory acidosis (10). If such an infection appears to be present, a suitable antibiotic agent should be given. It is our custom to employ parenteral crystalline penicillin or one of the broad-spectrum antibiotics, such as oral Aureomycin or Terramycin. Small daily doses of either penicillin, Terramycin or Aureomycin may be advisable for prophylaxis of respiratory-trace infections during the winter months, in patients prone to these conditions.

[3] Manufactured by V. Ray Bennett and Associates, Los Angeles, Calif.

If a patient with chronic cor pulmonale can be tided over a crisis, especially if his cardiac failure appears at the moment to be due largely to respiratory components, the physician will be rewarded and even amazed by the improvement, which may persist for months or even years. If the patient's failure is primarily cardiac, his outlook is usually not so favorable but, with careful management, he may do well for a surprising length of time.

The best treatment of chronic cor pulmonale is preventive, and should be attempted by treating any of the etiologic conditions as early and vigorously as possible. Among the useful aids in the interim management of the patient with chronic pulmonary emphysema are aminophylline, potassium iodide, bronchodilator aerosols, diaphragmatic breathing exercises, emphysema belts, pneumoperitoneum therapy and intermittent positive-pressure breathing with simultaneous bronchodilator sprays.

SUMMARY

The development of pulmonary arterial hypertension causes a progressive strain on the right ventricle which may ultimately lead to the development of chronic cor pulmonale. The combined effects of the primary pulmonary disorder and the secondary cardiac disorder are included in the term pulmono-cardiac disease. The prevention of pulmono-cardiac failure depends *largely* upon early recognition of the underlying pulmonary disease and the correction of the associated bronchial constriction, bronchial or parenchymal infections, pulmonary hypertension, hypoxia, or ventilatory defects. Chronic cor pulmonale may ultimately occur. Its management can be most rewarding, if the program outlined is vigorously carried out. Particular attention should be paid to the prevention and early recognition of the respiratory acidosis which may follow in the wake of the overzealous use of barbiturates, opiates and high concentrations of oxygen.

REFERENCES

1. Segal, M. S., and Dulfano, M. J.: Chronic Pulmonary Emphysema. Physiopathology and Treatment. New York, Grune & Stratton, 1953.
2. Spain, D. M., and Handler, B. J.: Chronic cor pulmonale. Sixty cases studied at necropsy, *Arch. Int. Med.* **77:** 37 (Jan.) 1946.
3. West, J. R.; Baldwin, E. de F.; Cournand, A., and Richard, D. W., Jr.: Physiopathological aspects of chronic pulmonary emphysema, *Am. J. Med.* **10:** 481 (Apr.) 1951.
4. Dexter, L.; Lewis, B. M.; Haynes, F. W.; Gorlin, R., and Houssay, E. J.: Chronic cor pulmonale without hypoxia, *Bull. New England M. Center* **14:** 69 (July) 1952.
5. Marks, M. O., and Zimmerman, H. A.: The roentgen and differential diagnosis of chronic cor pulmonale, *Am. J. Roentgenol.* **66:** 9 (July) 1951.
6. Bjurstedt, A. G. H.: Interacting of centrogenic and chemoreflex control of breathing during oxygen deficiency at rest, *Acta physiol. Scandinav.* **12:** 1 (Jan.) 1946.
7. Schmidt, C. F., and Comroe, J. H., Jr.: The role of the carotid and aortic bodies in the defense of the mammalian organism against oxygen lack, *Science* **92:** 510 (Sept.) 1940.
8. Segal, M. S.; Dulfano, M. J.; Herschfus, J. A., and Shanks, J. A.: Respiratory acidosis: pathogenesis and treatment, *Ann. Allergy* **11:** 206 (Mar.) 1953.

9. Barach, A. L.: Treatment of anoxia in clinical medicine, *Bull. New York Acad. Med.* **25:** 370 (June) 1950.
10. Stone, D. J.; Schwartz, A.; Newman, W.; Feltman, J. A., and Lovelock, F. J.: Precipitation by pulmonary infection of acute anoxia, cardiac failure and respiratory acidosis in chronic pulmonary disease, *Am. J. Med.* **14:** 14 (Jan.) 1953.
11. Barach, A. L.: Observations on methods of increasing resistance to oxygen poisoning in studies of accompanying physiological effects, *Am. J. Physiol.* **142:** 462 (Oct.) 1944.
12. Comroe, J. H., Jr.; Bahnson, E. R., and Coates, E. O., Jr.: Mental changes occurring in chronically anoxemic patients during oxygen therapy, *J.A.M.A.* **143:** 1044 (July) 1950.
13. Ferrer, M. I.; Harvey, R. M.; Cathcart, R. T.; Webster, C. A.; Richards, D. W., Jr., and Cournand, A.: Some effects of digoxin upon the heart and circulation in man; digoxin in chronic cor pulmonale, *Circulation* **1:** 161 (Feb.) 1950.
14. Zimmerman, H.: A study of the pulmonary circulation, *Dis. of Chest* **20:** 46 (July) 1951.
15. Miller, J. B., and Boyer, E. H.: A non-toxic detergent for aerosol use in dissolving viscid broncho-pulmonary secretions, *J. Pediat.* **40:** 767 (June) 1952.
16. Salomon, A.; Herschfus, J. A., and Segal, M. S.: Aerosols of pancreatic dornase in broncho-pulmonary disease, *Ann. Allergy* **12:** 71 (Jan.) 1954.
17. Dulfano, M. J.; Mack, F. X., and Segal, M. S.: Treatment of respiratory acidosis with N-allylnormorphine (Nalline), *New England J. Med.* **248:** 931 (May) 1953.
18. Salomon, A.; Marcus, P. S.; Herschfus, J. A., and Segal, M. S.: N-allylnormorphine in the treatment of morphine or demerol narcosis, *Am. J. Med.* (in press).
19. Eckenhoff, J. E.; Elder, J. D., and King, B. D.: The effect of N-allylnormorphine in the treatment of morphine or demerol narcosis, *Am. J. M. Sc.* **223:** 191 (Feb.) 1952.
20. Segal, M. S.; Salomon, A.; Dulfano, M. J., and Herschfus, J. A.: Pressure breathing. The use of intermittent positive pressure breathing in the inspiratory phase of respiration (IPPB/I), *New England J. Med.* **250:** 225 (Feb.) 1954.

THE MANAGEMENT OF EDEMA IN THE ELDERLY*

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The principles upon which the management of edema is based are not altered by the age of the patient, but it must be recognized that the aging process alone may introduce new factors into the clinical problems of edema which will influence the ultilization of those principles. It is the plan of this paper to review several of the fundamental facts of the etiology and pathogenesis of systemic fluid retention which must guide therapy and to indicate certain of the modifications which may be dictated by the patients' years.

CONGESTIVE HEART FAILURE

The most important of the disease processes accompanied by systemic edema is unquestionably congestive heart failure. Although the clinical manifestations of this syndrome are so familiar that their recital here would be repetitious, the concepts that have emerged from the uncounted clinical and experimental studies of cardiac decompensation which have flooded the medical literature during the last decade may be worthy of brief discussion.

Prior to this resurgence of interest, cardiac edema was generally believed to represent an accumulation of interstitial fluid that resulted from the elevated venous pressure accompanying a fall in cardiac output. The observations that weight gain might precede rather than follow an increase in venous pressure, that the phenomenon of congestive failure might appear in patients whose cardiac output is considerably greater than normal, and that in either of these instances there is a marked reduction in renal blood flow with a fall in glomerular filtration rate and in urinary excretion of sodium and water, have led to an increasing emphasis upon the kidney as the critical organ in the pathogenesis of cardiac edema. Most recently the adrenal cortex and the posterior pituitary, through their influence upon sodium and water metabolism, respectively, have been implicated as essential links in the chain of events which leads to the edema of congestive failure.

Out of this welter of often conflicting evidence has come a concept which may be partially acceptable to the adherents of each of these points of view and which has the additional advantage of placing the emphasis in cardiac decompensation upon the heart rather than upon the kidney, an endocrine gland, or an atom of sodium. It might be stated in the following way: when peripheral demand for blood exceeds the ability of the heart to meet that demand a series of events (involving first the heart but ultimately many organs) which includes an elevation of venous pressure, a deviation of blood away from the kidneys and an alteration in the renal mechanism for reabsorption of

* The study of edema by this laboratory has been supported through a grant from the Western New York State Heart Association.

TABLE 1

Relationship Between Peripheral Demand and Cardiac Output in Heart Failure

	Peripheral demand L/min.*	Cardiac output L/min.*
Normal		
At rest	5	5
With exercise	10	10
Arteriosclerotic heart disease (mild)		
At rest	5	5
With exercise	10	8
Arteriosclerotic heart disease (severe)		
At rest	5	4
Thyrotoxicosis	12	10
Anemia	12	10

* The figures used here are for illustration only and do not represent absolute values.

salt and water takes place, which leads to the appearance of a clinical state generally recognized as congestive heart failure.

Although such an elaborate description might appear needlessly academic and verbose, the words carry with them certain therapeutic implications which are of great importance to the patient suffering from dropsy. If a discrepancy between peripheral demand and cardiac output is the *sine qua non* of congestive failure, then it becomes imperative to discover the nature of that discrepancy if treatment is to be intelligent and effective. Table 1 shows several ways in which this discrepancy might occur.

The first example cited in Table 1 must certainly be the most frequent kind of heart failure to develop in an elderly population. In this category intrinsic myocardial or valvular disease results in a fall in cardiac output while peripheral demand remains constant. Logically the first and most important step in management should be an attempt to restore output to normal. Accomplishment of this end depends upon a single drug, *digitalis*, the pharmacologic effect of which is to increase the efficiency of myocardial contraction. The variety of preparations and the methods of administration of digitalis have multiplied many fold since Withering first described the use of foxglove in dropsy, but their effects are essentially the same. Despite strong opinions which have been expressed concerning the comparative efficacy and toxicity of whole leaf digitalis and its several glycosides (which masquerade under an imposing number of names) the choice of preparation is ultimately a matter of personal preference. When administered in adequate dosage each will produce a therapeutic effect and there is no convincing evidence that one is clearly superior to all others.

When digitalis in one of its forms does not achieve the expected relief of congestive failure, it is immediately apparent that the fundamental discrepancy between cardiac output and peripheral demand has not been resolved. In dealing with elderly patients three possible explanations for this failure stand out. In the first instance longstanding myocardial disease may have reduced cardiac reserve to the point where even the demands of a bed and chair existence exceed

the capacity of the heart to respond despite the additional support provided by digitalis. It is the kind of heart failure which requires the utmost ingenuity in utilizing the therapeutic weapons, which will be described shortly, aimed at the secondary metabolic changes of heart failure. Before dismissing the aged patient in this manner it is wise to consider the additional possibilities listed in Table 1, *i.e.*, that the problem may be mainly one of increased peripheral demand or decreased oxygen transport capacity rather than a functionally reduced cardiac output. Thyrotoxicosis is frequently masked in age and may manifest itself chiefly through congestive heart failure that reflects an increased cellular metabolism which exceeds the maximum output of a moderately arteriosclerotic myocardium. Anemia, a symptom of so many diseases which are common beyond middle age, can lead to an increase in the need for circulation of hemoglobin which outstrips the ability of the heart to respond. The failure of digitalis to produce a satisfactory remission of heart failure may be dependent upon these facts. Although this drug can often restore a reduced cardiac output to normal, it can rarely increase myocardial efficiency to a level which is adequate to meet such increased loads. Instead of digitalis, correction of the primary disorder by specific measures readily available is more likely to be accompanied by a gratifying improvement.

Although the imbalance between demand and output represents the primary physiologic defect in heart failure and the one which should receive the initial therapeutic attention, the metabolic disturbances which result from this discrepancy contribute significantly to the accumulation of edema. The most important of these defects is a profound alteration in salt and water metabolism.

Much has been written about the physiologic mechanism of this disorder but no final and generally acceptable explanation has yet been proposed. Reduced to its simplest terms, however, it is evident that the excretion of sodium (accomplished principally through the urine) is substantially less than the intake of this ion. When such a positive sodium balance exists, water too is retained, with the resultant appearance of edema. Whatever the mechanism, the corrective measure which immediately suggests itself is a reduction in sodium intake or the production of an increase in sodium excretion.

Reduction of dietary sodium is the most successful of the secondary measures for the relief of cardiac edema, but its accomplishment is sometimes at a considerable expense. The aged cardiac, habituated to salt by a lifetime of use may find the elimination of this seasoning an intolerable burden. If this is so, insistence upon such a policy will cause nutrition to suffer and an additional hazard to age may be induced by a well meaning physician. Several salt substitutes are presently available and may satisfy a significant number of the group which cannot abide saltless meals. They should not be overlooked. It was originally hoped that the cation exchange resins, by binding sodium in the gastro-intestinal tract, would make possible a more liberal dietary sodium content. It is unfortunate that further experience with these compounds has failed to confirm the hope, and although resin has a real usefulness in the treatment of congestive failure it is not as an agent which permits a less restricted diet.

At the other end of the spectrum are the measures available for producing an increase in sodium excretion. The *organic mercurials* are the most consistently useful in this regard. It is worth recalling that the effectiveness of these compounds depends upon the amount of ionized mercury which reaches the renal tubule. Whatever the preparation or route of administration, comparable amounts of the ion will produce comparable diuretic effects. Failure to deliver therapeutic quantities of mercury to the end organ is one of the common causes of mercurial failure. It is for this reason that larger total amounts of mercury must be given orally than parenterally, since the gastro-intestinal tract absorbs mercury poorly. A usually effective parenteral dose may be ineffective for a similar reason if it is given into an edematous buttock where absorption is poor. To those who insure an adequate delivery of mercury to the tubule by intravenous administration a word of caution is in order. The most serious complication of mercurial therapy is the development of ventricular fibrillation, an aberrant rhythm that is produced only by sudden high concentrations of mercury. Such concentrations can be achieved only when the drug is introduced directly into a vein.

One further complication of mercurial diuresis is worthy of mention. The most prominent pharmacologic effect of this drug appears to be a disturbance in the tubular reabsorption of chloride ions. Accumulation of chloride ions in the tubular urine requires the presence of an equivalent number of cations. Sodium is the principal cation bound and excreted in this fashion. However another cation, potassium, may also be excreted in significant amount following the administration of a mercurial diuretic. In the fully digitalized patient reduction of extracellular potassium concentration may be accompanied by the development of digitalis intoxication. Such intoxication is the result of potassium deficit, not digitalis excess. It should be treated first by potassium supplements rather than digitalis elimination.

The *cation exchange resins* may also be of great benefit in achieving a negative sodium balance. As pointed out earlier, resin will accomplish this result only when it is administered with a low salt diet. When this condition is met, the fecal excretion invariably exceeds the dietary sodium. The patient may find this small comfort, however, for resin frequently leads to gastro-intestinal disturbances which may be very distressing. To the aged patient the most annoying is usually constipation, and the flabby abdominal musculature of the elderly subject may be unfit for the task of expelling the hard stool. Although this side effect of resin therapy is not life threatening, another less frequent complication may pose such a threat. The binding of sodium in the gastro-intestinal tract limits the fixed cation available for urinary excretion with the anionic end products of metabolism. The renal tubule is thus placed under a maximal stress to produce ammonia as a substitute for sodium. The normal kidney responds promptly and efficiently to this stimulus but a diseased kidney reacts in a laggard fashion. The result may be the accumulation of anions in the extracellular fluid, with the development of a severe systemic acidosis. Since the aging kidney is so often functionally damaged, this danger of resin therapy

should be kept firmly in mind when dealing with elderly patients and guarded against by frequent determinations of carbon dioxide combining power. Because of these potential problems it is probably wise to reserve the cation exchange resin for the treatment of edema which is resistant to other measures and then to use it intermittently. Intermittent administration of resin has the additional advantage of providing maximal potentiation of mercurial diuresis.

HEPATIC CIRRHOSIS

Of the other causes of systemic edema only two will be considered briefly. Hepatic cirrhosis is difficult to discuss logically, for understanding of the physiologic alterations which lead to fluid retention in this disease is even more sketchy than is the case with congestive heart failure. Traditionally the edema associated with cirrhosis has been attributed to the two prominent manifestations of hepatic disease which may lead to an abnormal distribution of fluid across the capillary membrane—reduced serum concentration of albumin and portal hypertension. Correction of these abnormalities would seem a reasonable point of departure in therapy.

The failure of albumin synthesis which results from extensive hepato-cellular disease is not a quickly reversible process. The intravenous administration of salt-free human albumin has been suggested, to achieve a rapid restoration of normal serum protein concentration with expectation of an attendant reduction in edema. Therapeutically this agent has been disappointing, for few cirrhotic patients respond with a diuresis and none shows any persistent elevation of serum albumin for more than a few hours after the infusion. In the elderly subject such therapy is not without its dangers. The increase in serum oncotic pressure which attends the administration of albumin may lead to an increase in circulating plasma volume sufficient to embarrass an arteriosclerotic myocardium, with a resulting acute left ventricular failure. Far more reliable as a means of restoring albumin to its normal level, is the empiric provision of a diet high in calories and vitamins, which provides protein in excess of basal needs. It is not entirely clear why such therapy is effective, but of all methods available it is most likely to be successful in restoring many hepatic functions, including albumin synthesis, to normal. As these functions are restored fluid retention may subside, but without close correlation with serum protein concentration.

The architectural change which is produced by hepatic cirrhosis and which causes portal hypertension cannot be expected to vanish with diet therapy. If ascites persists in spite of adequate dietary treatment it is often believed that local hypertension in the portal bed is the cause. From this point of view surgical anastomosis of the portal and systemic venous systems with a reduction in portal vein pressure seemed a hopeful approach to the treatment of persistent ascites in cirrhosis. Although such surgery has been successful in reducing portal pressure it has not been successful in relieving abdominal accumulations of fluid and, on the basis of experience to date, it is generally accepted that the portal-systemic venous shunt should be reserved for the treatment of the bleeding complications of portal hypertension.

Equally important in the development of fluid retention in cirrhosis are the metabolic disturbances which bear a close resemblance to those found in heart failure—the tenacious retention of sodium, which is accompanied by the retention of water. There are few data available which can provide any generally acceptable explanation of such defects. Current speculation centers upon the endocrine controls of water and salt regulation and the influence of impaired hepatic function on the utilization of adrenocortical and posterior pituitary hormones. As yet no definitive answer has been forthcoming and no therapeutic aid provided by extensive and continuing studies of these phenomena. Instead the physician faced with the problem must depend upon empiric evidence that reduction of sodium intake or increase of sodium excretion provides the most hopeful avenue of approach. The methods described in the discussion of cardiac decompensation apply equally to cirrhosis, but the results are seldom as gratifying.

In using cation exchange resins to produce an increase of sodium excretion in the cirrhotic patient, one additional caution must be cited. Among other hepatic functions which may be impaired in diffuse hepato-cellular disease is the ability to convert ammonia to urea. If resin in an ammonium form is used, the absorption of this radical may lead to accumulation of ammonia in the plasma, with the appearance of a syndrome which is usually indistinguishable from hepatic coma. Although this state is generally reversible, it can be avoided by the use of resin in the hydrogen form.

CHRONIC WASTING DISEASE

Finally a word should be written about the edema associated with chronic wasting disease, a kind of fluid retention which is not infrequent among elderly patients. Unfortunately it is difficult to categorize this condition or to discuss it intelligently, for information concerning etiology and pathogenesis is meager indeed.

In the past there was a general belief that this represented a condition not unlike famine edema which may result from hypoproteinemia or B avitaminosis; but it has become increasingly evident that few of these patients exhibit significant depression of serum albumin concentration and although many show multiple vitamin deficiencies the incidence of frank beri-beri is rare. Moreover, specific measures designed to correct plasma protein deficiency or to replace vitamin deficits do not lead regularly to remission of symptoms and signs.

It is striking that in this situation again the curious disturbance of water and salt metabolism, previously described in relation to congestive heart failure and hepatic cirrhosis, may also be demonstrated. The ultimate nature of the responsible mechanism is even less clear here than in other diseases mentioned. This lack of understanding compounds the difficulty of dealing with the problem. Unless the primary disease is reversible, the only therapeutic suggestion which has merit at the present time is again the creation of a negative sodium balance by the means already described. It is only fair to point out, however, that in this instance these measures often seem like idle gestures.

SUMMARY

The examples cited represent a physiologic approach to edema which has proved useful in an evaluation of the therapeutic needs of individual patients. It seems not unlikely that specific therapeutic measures may be modified as additional information concerning the pathogenesis of these disease processes is revealed. In the meantime the elderly patient, like the youth, will benefit from the systematic application of currently acceptable concepts.

COMPLICATIONS OF DIABETES IN OLDER PATIENTS

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Some of the characteristics of diabetes mellitus in patients over 60 years of age have been discussed in a recent report (1). Obviously, in the older age groups many conditions affect the primary disease and in some cases even overshadow it.

In order to separate so-called "complications" from symptoms inherent to the diabetes *per se*, a clearer definition of terms is necessary. According to Webster a complication is a "disease or adventitious condition co-existent with and modifying a primary disease." Thus one must exclude manifestations like acidosis and coma, which result directly from the diabetic state. These may be referred to more appropriately as "components" or "constituents" of the disease process. Other conditions which fall into this latter category are retinopathy and Kimmelstiel-Wilson's disease (nephropathy). Although the exact etiologic relationship of neuropathy to diabetes is still controversial, I am in accord with those who view this entity as an integral (component) part of the disturbed metabolic state. Evidence in support of the interrelationship of retinopathy, glomerulosclerosis (Kimmelstiel-Wilson's disease) and the neuropathies to diabetes has been presented previously (2, 3). Suffice it to say, these conditions occur in the elderly diabetic to approximately the same extent as in diabetics as a whole. For instance, retinopathy was found in 25 per cent of the patients over 60 years of age, and evidence of neuropathy in more than 50 per cent, only a slightly lower incidence than in diabetics in general. I classify liver enlargement as part of the symptom-complex of unregulated diabetes (4), except in well controlled diabetic patients and those with lipotropic deficiency, in whom hepatomegaly may be considered a complication. The conditions just mentioned, being components and not true complications, have been discussed at length elsewhere and will not be included herein.

Tabulation of the various conditions associated with diabetes mellitus (Table 1) reveals that most older patients have one or more complications. Some are more significant than others; only the more important ones will be discussed here. Whereas some complications, *e.g.*, impaired vision, or peptic ulcer, may hamper control of the diabetes, in some instances diabetes may accelerate the course of the accompanying disease. The more important complications observed in elderly diabetics are, in the order of their frequency: (A) atherosclerosis, (B) ophthalmologic conditions (excluding retinopathy), (C) benign prostatic hypertrophy, and (D) chronic infections (Table 1).

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TABLE 1

Associated Conditions in Aged Diabetic Patients

	No. of Cases	
1. Atherosclerosis		57
A. Proximal	51	
B. Peripheral	31	
1) Amputations	6	
Male	5	
Female	1	
2) Diabetic ulcers	6	
C. Coronary	22	
Male	14	
Female	8	
1) Infarcts (all males)	4	
D. Cerebral	7	
1) Strokes	6	
Male	5	
Female	1	
2. Ophthalmologic conditions (excluding retinopathy)		21
A. Cataract	16	
Male	10	
Female	6	
B. Corneal arcus	10	
3. Prostatic hypertrophy		12
4. Chronic infection		11
A. Bronchitis	4	
B. Otitis media	3	
C. Ischio-rectal abscess	2	
D. Gingivitis	1	
E. Conjunctivitis	1	
F. Vaginitis	1	
5. Hernia		10
A. Inguinal	9	
B. Hiatus	2	
6. Varicose veins		9
7. Hypertrophic arthritis		8
8. Senile emphysema		8
9. Hypertension		7
10. Kidney diseases		4
11. Carcinoma		4
A. Rectum	1	
B. Bladder	1	
C. Cervix	1	
D. Intestine	1	
12. Alcoholism		3
13. Peptic ulcer		3
A. Resected	1	
14. Edema		2
15. Psychoneurosis		2
16. Rectocele		2
17. Cystocele		2
18. Ecchymoses		2
19. Parkinsonism		1
20. Absent testis		1
21. Kyphosis		1
22. Colloid goiter		1
23. Herpes zoster		1
24. Cholecystectomy		1

A. ATHEROSCLEROSIS

In a recent survey of 59 patients, in whom diabetes mellitus was discovered after they had attained 60 years of age, atherosclerosis was detectable clinically in 57, or 96 per cent of the cases (1). Coronary atherosclerosis was diagnosed by physical examination and/or electrocardiography in 22 patients, 14 males and 8 females; 4 patients (males) had had myocardial infarctions.

In the newer classification of the arterial diseases atherosclerosis is included among the "degenerative arteriopathies" (5). As Wolffe points out, the term "arteriosclerosis" lacks clinical specificity. In degenerative arteriopathy there is gradual deterioration of the cellular components of the vessel wall typified by 1) atherosclerosis, 2) medial sclerosis and 3) arterionecrosis.

Of the three, atherosclerosis, a leading cause of chronic disability and death in this country, is the most important arteriopathy encountered during and after middle age. Over 30 per cent of persons over 60 years of age die principally as a result of atherosclerosis (6). Atherosclerosis affects not only the large elastic arteries such as the aorta and the great vessels but also smaller arteries in vital areas (heart, brain). Coronary atherosclerosis is simply a particular manifestation of generalized atherosclerosis, and gives rise to symptoms only as the blood supply of the heart muscle is curtailed. In a sense, angina pectoris, coronary thrombosis and so-called coronary insufficiency are not clinical entities, but merely steps in the slow evolution of atheromatosis of the coronary arteries.

By and large, atherosclerosis in the diabetic does not differ from that found in nondiabetics. The outstanding feature of diabetics' morbidity and mortality rates is the unusual frequency of advanced occlusive vascular lesions involving chiefly the coronary and leg arteries. In the preinsulin era, coma was the most common immediate cause of death in diabetes (64 per cent). As a cause of death, it has been replaced by atherosclerosis and its sequelae, including coronary thrombosis. The frequency of coronary atherosclerosis has increased greatly as other causes of death have diminished so that, at present, the incidence of coronary occlusion and myocardial infarction in diabetes is distressingly high, directly accounting for about half of all deaths. Indeed, occlusion of atherosclerotic coronary arteries has far outstripped other causes of death in diabetic patients in recent years and the incidence of angina pectoris and coronary insufficiency has increased proportionately. Millard and Root (7) reported demonstrable coronary atherosclerosis in 108 of 110 autopsies on diabetics at the New England Deaconess Hospital in the period 1940–1946. The frequency of gangrene of the lower extremities is too well known to require emphasis.

Root, Bland *et al.* (8) have shown that diabetics, and especially diabetic women between the ages of 51 and 80 (from postmortem evidence), have a much higher incidence of severe myocardial infarcts than nondiabetics. E. T. Bell (6) reported that in 1,214 diabetic autopsies there was evidence that coronary atherosclerosis *caused death* twice as frequently in male diabetics as in female nondiabetics and three times as often in diabetic as in nondiabetic females. Gangrene was 40 times as frequent in diabetic as in nondiabetic females past 50 years of age. From this it would appear that clinical obstruction of the

blood vessels, at least in the coronary and leg arteries, develops more frequently, at an earlier age and in more severe form in diabetics than in nondiabetics.

One should remember that the most common age for the development of diabetes is around 50 years, a period when susceptibility to atherosclerosis is also great. Provided he lives long enough, a diabetic will die of, or with atherosclerosis, just as any other individual. Bell (6) found that, whereas 54 per cent of diabetics over 40 years of age die of some form of atherosclerosis, 29 per cent of nondiabetics die in the same way. Out of every 10 diabetic patients who died of coronary atherosclerosis 7 could have been expected to die of this cause had they not had diabetes. In other words, even under ideal conditions, one cannot hope to lower the mortality rate of diabetics below 29 per cent. Even if all other complications and causes of death were eliminated in diabetics, 100 per cent would then die of atherosclerosis. Obviously, in the older groups there are many vascular deaths now attributable to diabetes. However, conclusive experimental evidence that diabetes enhances atherosclerosis is still lacking.

Joslin (9) brings out an interesting fact, namely, that angina pectoris occurs most frequently in the seventh decade, in which ten-year cases of diabetes are most common. He reasons that several-years' duration of diabetes is an important factor in the etiology of angina pectoris in this group, since diabetes develops most frequently at the age of 51.

1. *Clinical criteria for the diagnosis of atherosclerosis.* In the earliest stages atheromata are present for some time without producing obvious clinical phenomena. The clinical signs of advanced atherosclerosis depend upon which organ is the seat of the occlusion, *e.g.*, cerebrum, heart, aorta (dissection, rupture, aneurysm), or mesenteric artery. Unequivocal evidence of coronary atherosclerosis is manifested by electrocardiographic changes, classic anginal pain and, in the lower extremities, by impairment of blood flow. In many persons with severe atherosclerosis, probably cardiac enlargement (as determined by fluoroscopy) is also indicative of coronary artery sclerosis.

The living patient has been investigated with increasing care in recent years for signs of atherosclerosis. My associates and I (10) found the following clinical criteria to be the most reliable: (a) positive results with the carotid-sinus pressure test, (b) palpable subclavian pulses, (c) clinical signs of impaired peripheral blood flow, and (d) calcification of the aortic arch or the abdominal aorta as observed in x-ray films.

By means of *roentgenographic examination* atherosclerosis may be detected before obliteration occurs. Pathologically, the intimal lesions of atherosclerosis have a predilection for the lower abdominal and posterior thoracic aortic areas (Aschoff). Likewise, roentgenographically, calcified plaques are frequently demonstrable in the lower abdominal aorta just above the bifurcation, and in the thoracic aorta.

A calcific aortic plaque, identified radiologically, constitutes most reliable evidence of atherosclerosis (10, 11). In the thoracic aorta atherosclerosis is most often seen in the knob. We (10) found the incidence of calcific plaques to be 41.5 per cent in a series of 89 diabetics. Sufficient emphasis has not been given to

this finding in the current literature. The demonstration of calcified plaques in the aortic knob in the conventional chest film is valuable evidence of atherosclerosis, and there is no better means of demonstrating this condition. Another significant roentgenographic sign is a widening of the supracardiac shadow owing to uncoiling of the upper part of the thoracic aorta, with veiling which extends from the aortic knob to the pulmonic conus. Since there is a fairly close parallelism between the degrees of coronary, cerebral and aortic sclerosis, atherosclerosis in the upper aorta (proximal atherosclerosis) is generally a good index of the process in the coronary and cerebral vessels, although discrepancies may be found.

In estimating the peripheral blood flow it is not always easy to evaluate the dorsalis pedis and posterior tibial pulses by tactile sense alone. The presence of trophic changes, such as dry scaly skin, thickened nails, scarring, or shrinking of the digits, points to ischemia. By passing the hand downward from the patient's thigh a change in skin temperature may be detected. However, in my personal experience, oscillometry provides the best quantitative evidence of the degree of occlusion and the level at which larger vessels are occluded. More important is the collateral circulation, in the estimation of which skin temperature is an important factor.

2. *Medial (Mönckeberg's) arteriosclerosis.* This second variety of degenerative arteriopathy consists of diffusely distributed calcium deposits in the media of muscular arteries, such as the femoral (lower two thirds), tibial, pelvic, radial and temporal. The calcium may be deposited in rings giving a "gooseneck" appearance. The lesions begin early in life and are not limited to older persons, although probably it is part of the aging process. Since medial calcification is readily demonstrated by roentgenogram, the physician as well as the patient should be aware of it, though not needlessly concerned. Medial arteriosclerosis is asymptomatic unless associated with atherosclerosis.

3. *Other types of arteriosclerosis.* Bell (6) found *arteriolar sclerosis* of the renal vessels more than four times as frequently in diabetics over 50 years of age than in controls of comparable age. Productive or *hyperplastic arteriopathy* of senescence, which is due to fibrosis of the intima and media with thickening and elongation of the vessel, is not symptom-producing, though it may be found in combination with other significant arteriopathies.

4. *Treatment—Diet.* Reliable data as to the effect of diet on human atherosclerosis are both meager and unsatisfactory. Available evidence suggests that atherosclerosis may be affected by limitation of vegetable and animal fats and probably cholesterol (13), as well as total caloric intake. Restriction of body weight to the ideal weight level is strongly advised in the obese. The question is, should a patient of normal weight who has clinical evidence of atherosclerosis have his diet sharply curtailed?

Some progress in the treatment of peripheral atherosclerosis has been made as a result of the lowered incidence of burns from hot water bottles. Provided that trauma and cracks in the skin of the feet can be avoided, infection and gangrene are preventable. Judging by the prevalence of gangrene due to known

trauma, it is obvious that diabetic patients in general have not been impressed sufficiently with the importance of foot care and protection. Every diabetic should be forewarned against using patent medicines and trimming corns and calluses with razor blades. Trichophytosis, an innocuous disease in an individual with adequate circulation, may cause a major disorder in a diabetic with a blister or crack in the skin. Woolen socks or a warm blanket are recommended to keep the feet warm in place of electric pads, hot water bottles or hot foot baths. The patient should wear shoes at all times both in the home and outdoors, in order to avoid splinters, tacks or stray pieces of glass. Factory workers should be instructed to wear safety shoes with steel toes. The texture of the skin can be kept soft with lanolin or vaseline after daily cleansing and thorough drying of the feet.

Surgeons are becoming more and more conservative in the treatment of the gangrenous foot in diabetes mellitus. They have learned that many patients who refused an amputation which was deemed necessary have done exceptionally well on a more conservative basis. I subscribe to the conservative approach.

B. OCULAR COMPLICATIONS

Excluding diabetic retinopathy, which is classified here as a constitutent of diabetes, cataract is the most important ocular finding in older diabetic patients. In 59 patients in whom diabetes began after the age of 60 years, cataract was noted in 16, an incidence over 25 per cent; this is a higher incidence than Barach found in 317 such patients (10 per cent). Cataract is usually the simple senile type, with no proven relationship to the diabetes. In fact, in Waite and Beetham's (13) large, careful study reported in 1935, simple cataract actually was found more frequently in nondiabetics than in diabetics.

The status of so-called arcus senilis, observed in 10 of the 59 patients examined, has been reappraised recently, principally by Adlersberg. Whereas this entity has been accepted heretofore without question as a sign of the aging process, Adlersberg noted its association with abnormal cholesterol metabolism, and termed it "corneal arcus."

C. BENIGN PROSTATIC HYPERTROPHY

The high incidence of benign prostatic hypertrophy in 12 of the 36 male diabetic patients over 60 years of age ought not to be surprising, nor the fact that many of them had undergone prostatic surgery. However, in view of the similarity and possible confusion of the symptoms of prostatism with those of diabetes, it is important to differentiate the two conditions. The hazards of prostatic obstruction, urinary retention and uremia are well known. On the other hand, the potential untoward effects of poorly controlled diabetes in patients who are to undergo prostatic surgery are equally important.

D. CHRONIC INFECTIONS

The existence of a chronic infection in 11 of our 59 patients (approximately 1 out of 5) with onset of their diabetes after 60 years of age, deserves some com-

ment. The question arises as to whether the coexistent diabetes may have had a bearing upon the chronicity of these infections. Upon closer analysis of the nature of the chronic infections observed (Table 1), it is doubtful that the diabetes exerts any influence. Chronic bronchitis and otitis media, for example, are frequent occurrences in nondiabetic patients also. Conversely, the state of diabetic control seemingly was undisturbed by the chronic infection.

The remaining conditions listed in Table 1 do not appear to have any influence on the diabetes of older individuals and, in turn, are unaffected by the diabetes. They simply represent a cross section of the many diseases to which aged persons in general are susceptible.

SUMMARY

1. The complications of diabetes mellitus reviewed in this paper are *complications* in the truest sense of the word. Conditions such as acidosis, retinopathy, glomerulosclerosis, neuropathy and hepatomegaly, which are considered by many as inherent to diabetes, are classed as components and not considered here.

2. The most significant complications found in elderly diabetic patients are atherosclerosis, ophthalmologic conditions, benign prostatic hypertrophy and chronic infections.

3. The generic concept of arteriosclerosis lacks specificity and is no longer acceptable. a) The types of vascular disease especially accelerated by diabetes are coronary atherosclerosis, gangrene and renal arteriosclerosis. b) Atherosclerosis was detected in 96 per cent of 59 patients who were over 60 years of age at the onset of diabetes. c) The best clinical criteria for the early detection of atherosclerosis are reviewed briefly. d) The importance of prophylactic foot care in the prevention of gangrene is stressed.

4. The interrelationship of diabetes and the other principal complications—ophthalmologic manifestations, benign prostatic hypertrophy and chronic infections—is discussed.

REFERENCES

1. Goodman, J. I., and Goldberg, L. B.: Diabetes mellitus in the aged, *Ohio State M. J.* **49:** 981 (Nov.) 1953.
2. Goodman, J. I.: The specific vascular lesions of diabetes mellitus. II. retinopathy and intercapillary glomerulosclerosis, *Am. J. Ophth.* **36:** 957 (July) 1953.
3. Goodman, J. I.; Baumoel, S.; Frankel, L.; Marcus, L. J., and Wassermann, S.: The Diabetic Neuropathies. Springfield, Illinois, Charles C Thomas, Publisher, 1953.
4. Goodman, J. I.: Hepatomegaly and diabetes mellitus, *Ann. Int. Med.* **39:** 1077 (Nov.) 1953.
5. Wolffe, J. B.: The role of arterial diseases in clinical medicine: some comments on classification of arteriopathies, *J. Am. Geriat. Soc.* **1:** 157 (Mar.) 1953.
6. Bell, E. T.: A postmortem study of vascular disease in diabetics, *Arch. Path.* **53:** 444 (May) 1952.
7. Millard, E. B., and Root, H. F.: Degenerative vascular lesions and diabetes mellitus, *Am. J. Digest. Dis.* **15:** 41 (Feb.) 1948.
8. Root, H. F.; Bland, E. F.; Gordon, N. H., and White, R. D.: Coronary arteriosclerosis in diabetes mellitus, *J.A.M.A.* **113:** 27 (July 1) 1939.

9. JOSLIN, E. P.; ROOT, H. F.; WHITE, P., AND MARBLE, A.: Treatment of Diabetes Mellitus, ed. 9. Philadelphia, Lea & Febiger, 1952.
10. GOODMAN, J. I.; WASSERMANN, S.; MARCUS, L. J., AND FRANKEL, L.: A study of atherosclerosis in a group of diabetic patients, *Am. J. M. Sc.* **220:** 30 (July) 1950.
11. FRAY, W. W., AND GREEN, J. H.: Structural changes of the aorta due to arteriosclerosis, as observed roentgenologically, *New York State J. Med.* **33:** 694 (June) 1933.
12. HILDRETH, E. A.; HILDRETH, D. M., AND MELLINKOFF, S. M.: Principles of a low fat diet, *Circulation* **4:** 899 (Dec.) 1951.
13. WAITE, J. H., AND BEETHAM, W. P.: The visual mechanism in diabetes mellitus: a comparative study of 2,002 diabetics, and 457 nondiabetics for control, *New England J. Med.* **212:** 367 (Feb.); 429 (Mar.) 1935.

METHONIUM COMPOUNDS IN THE TREATMENT OF HYPERTENSION*

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A mild sensation was recently caused by the announcement in screeching newspaper headlines of the allegedly remarkable effect of methonium compounds in the treatment of hypertension. Extravagant lay interpretation, based on clinical reports by a few physicians, brought on an avalanche of inquiries regarding the April miracle. Although the ideal medicament is, unfortunately, still elusive it can be said of methonium compounds that they do show definite potentialities.

As far back as 1928, Fahr (1) estimated that more than 140,000 deaths annually may be attributed to hypertensive cardiovascular disease in the United States alone. Many times that number of persons are handicapped invalids as the result of hypertension.

Every clinician is aware that hypertension is a symptom and not a disease. It appears in a few known, and in many idiopathic morbid conditions. Specific measures apply to only a relatively small number of patients with hypertension. We studied 500 consecutive patients who presented themselves for examination and found that of 3 with pheochromocytoma only 1 was cured following surgical intervention, and of 6 with so-called "unilateral surgical renal disease" only 2 were completely relieved of hypertension for more than three years following surgery. Cured hypertensive patients in our series, therefore, constituted little better than 0.5 per cent.

Convincingly demonstrated, unilateral renal ischemia may be an indication for surgical removal of the involved kidney, if associated with persistent high blood pressure (2, 3). Yet we all recall that only a score or more years ago a great number of nephrectomies were performed upon the discovery of even asymptomatic unilateral nephropathies, solely for the relief of hypertension. The results were disappointing.

The surgical approach to the treatment of hypertension was also received with too much unwarranted enthusiasm but, in retrospect, extensive sympathectomies have added comfort and years to the lives of some hypertensive persons (4, 5).

The multiplicity of factors producing hypertension is still beyond our present state of knowledge. So far, all we may conjecture is that normal arterioles are

* Read at the Annual Meeting of the American Therapeutic Society, Chicago, Ill., June 5-8, 1952.

vulnerable to countless irritants which may set in motion the pathogenesis of hypertension. This symptom-complex gains expression through both components of the autonomic nervous system, *i.e.*, the sympathetic and parasympathetic pathways and their endocrine and metabolic counterparts.

Until we are able to discern specific etiologic factors in hypertension our search may also be directed to the discovery of remedies which should at least block, buffer or act as "shock absorbers" for this trigger mechanism. There is more than suggestive evidence that the methonium compounds may be one of the first groups of drugs to act in such a capacity. Their action on the autonomic nervous system may explain the beneficial results reported following the use of methonium in such varied diseases as gastric ulcers, obliterative peripheral vascular disease and hypertension.

METHONIUM COMPOUNDS

It may be of interest to state briefly how the methonium compounds were introduced in the treatment of hypertension.

Decamethonium has been employed as a substitute for curare, because of its relaxing effects during abdominal surgery (6); but it had an undesirable effect on respiration, which limited its use. Pentamethonium and hexamethonium compounds were investigated as substitutes and theoretically as antidotes for decamethonium (7). It was found that a "side-effect" was the reduction of blood pressure to a pronounced degree. These drugs are hypotensive to such an extent that our English colleagues use them in what they term "bloodless surgery" on the head and neck.

Such chemical compounds are of a series of quaternary ammonium salts of the general formula $(CH_3)\ N + (CH_2)_n\ N - (CH_3)_3\ 2X$, in which n represents the number of CH_2 groups and X equals the acid radical of the salt. Thus C_5 is the pentane derivative, in which n equals 5; and C_6 is the hexane derivative, in which n equals 6. These compounds are, therefore, referred to as pentamethonium and hexamethonium compounds, respectively.

All methonium salts are, as a rule, colorless, water-soluble crystalline powders of acid reaction, having a definite melting point. They may be readily sterilized without loss of effectiveness. It has been found by other investigators that hexamethonium salts are somewhat interchangeable with the penta salts, but the former seem slightly more effective in lowering blood pressure.

The action of the methonium compounds in hypertension bears a similarity to the action of tetraethyl ammonium salts but differs in the direction of greater potency, slower and longer duration of action and less toxicity (8).

Our experience has been limited to hexamethonium bromide, chloride, iodide and bitartrate and we shall confine our remarks to these preparations interchangeably.

Contrary to other investigators, we found that to some extent methonium salts inhibit the effect of epinephrine as well as does Desympatone. We consider the latter to be the parasympathetic hormone and humoral epinephrine antagonist (9).

The methonium compounds act as sympathetic as well as parasympathetic blocking agents. Because of the latter properties, their use in hypertension has been referred to as "medical sympathectomy." We regard them as "autonomic buffers" or "shock absorbers."

The present report contains some of the facts which I gleaned through the courtesy of Prof. M. J. Rosenheim of the University College Hospital in London, who was gracious enough to review with me some of his significant clinical data. I was impressed by his English conservatism and the thoroughness with which the drug was investigated by the members of his staff (10).

In November 1951, one of us (JBW) reported before the Annual Meeting of the American Society for the Study of Arteriosclerosis in Chicago, the results in 80 hypertensive patients treated with methonium (11). The present report is based on further observations on the same patients and an additional group of 70. In the latter group the method of administration of the drug was changed, based on the experience gained with its use.

In the first series of patients, we began the administration of methonium parenterally, since it was the only preparation made available to us from England. At a later date, as soon as we obtained the oral preparation, we substituted it whenever possible.

Susceptibility to the parenteral use of the drug is unpredictable and at times alarming. Therefore, administration should be started cautiously with a test dose of 5 mg. If at all possible the patient should be hospitalized during parenteral administration of methonium compounds, until the ideal dose has been established.

In most cases the lowering of the blood pressure is most marked when the patient assumes the erect position. If this is not taken into consideration when the maintenance dose of the drug is being established, dizziness and fainting may result when these patients stand for any length of time. The incidents often alarm the patient as well as the inexperienced members of the staff. Coronary vascular thrombosis has been reported by some investigators as the result of the precipitous and sometimes unpredictable lowering of the blood pressure following parenteral administration of methonium compounds.

In the past, we considered any suggestive evidence of thrombosis within the retinal, coronary, and particularly the cerebral vessels, as a contraindication to the use of these compounds. This precaution was in agreement with the findings of other investigators (12).

However, in the light of more recent observations, we consider the judicious use of the drug warranted in some cases of vascular thrombosis, provided the elevation in blood pressure *per se* constitutes a danger to the patient's welfare. The patient's condition must be considered in its entirety, rather than the blood pressure readings alone. The blood pressure should be lowered to a safe degree rather than to a desired objective level.

Many unexpected good results in our series can only be attributed to the use of methonium. The following case report is one of many, and may serve as an illustration.

Case report

B. H., a male white physician, aged 56 years, was examined by Dr. N. W. Winkelman of Philadelphia on September 28, 1951. At that time he gave a history of weakness of the right side of the body, with some difficulty in speech four years previously. The neurologic diagnosis at that time was "thrombosis of the Sylvian artery." Two years prior to this examination he had commenced to experience vertigo, had tended to fall to the right side and had diplopia. At that time a diagnosis of another cerebral lesion was made. For the previous few days the patient had not been feeling well. He went to bed early but in the morning he felt sick and had blurring of vision associated with double vision. The gait has been somewhat weak, more so on the left side of the body.

Pertinent neurologic findings: The patient was short, obese, not too alert and was able to give only a very sketchy history, particularly for a physician. The pupils were equal, regular and responded promptly to light; convergence was good. There seemed to be bilateral nystagmus. There was some restriction in lateral movements of the eyeballs and there was no doubt about the fact that the disturbance of the ocular movements was the result of a central lesion. The blood pressure was 190/120. The reflexes in the upper limbs were distinctly hyperactive, with a predominance of the left side. A Hoffman sign could be elicited bilaterally, but was particularly noticeable on the left side. There was no sensory disturbance. The reflexes in the lower limbs were distinctly hyperactive, with a predominance of the right side. There was no Babinski on either side. Vibratory sense was intact bilaterally. There was a definite memory loss.

Diagnosis: Hypertensive cerebrovascular disease with numerous small occlusive lesions. The entire symptomatology could be explained on the basis of these lesions, with recent episodes in the brain stem which occasioned a disturbance of ocular movements with diplopia.

Subsequently the doctor was admitted to the Valley Forge Heart Institute in Fairview Village, Pa., for methonium therapy. He received 25 mg. of methonium bromide parenterally every twelve hours until his blood pressure was stabilized. He was then given 250 mg. of methonium bromide orally in addition to rutin-methyl-glucamine 100 mg. (13, 14, 15) and ⅛ grain of phenobarbital three times daily.

Re-examination by Dr. N. W. Winkelman on October 26, 1951: The patient stated that upon inception of therapy the weakness, dizziness and diplopia disappeared. No abnormalities of the cranial nerves were found at this time. Bilateral nystagmus and restriction of the lateral movements of the eyeballs had disappeared. The reflexes were still slightly hyperactive although equal on both sides, as was the Hoffman sign. The reflexes in the lower limbs were still somewhat overactive but equal in both limbs. The blood pressure was 160/80.

Comment: There was a startling improvement in this patient's neurologic signs in a comparatively short period of time—an improvement which usually does not occur even under the most favorable circumstances.

The doctor returned to the practice of medicine. He continued to take the oral combination of methonium, rutin-methyl-glucamine and phenobarbital every eight hours around the clock, in addition to 25 mg. of methonium parenterally every twelve hours. His blood pressure readings varied between 135/80 standing and 150/95 sitting.

PRESENT METHOD OF ADMINISTRATION

Since there is no particular rush in the reduction of blood pressure, we found it best to start the administration of these compounds orally. It seems to matter very little which one of the methonium salts is used. However, if large doses are needed it is best not to use methonium bromide because of possible bromism.

Only 30 per cent of our last series of 70 cases needed hospitalization. A test dose of 250 mg. of methonium chloride or bromide or 350 mg. of methonium

bitartrate is given orally, preferably on an empty stomach or before a meal, to encourage absorption. The patient is observed very carefully and blood pressure readings are taken every fifteen to thirty minutes for at least two hours. This is carried out mainly as a tolerance test rather than for the effect in lowering blood pressure. If no untoward symptoms occur, the patient is instructed to repeat the dose every eight hours around the clock for a few days; then it may be taken as warranted. In most instances no reduction in blood pressure is noted for days and even weeks following the inception of graduated oral therapy.

The patient is instructed to skip a dose or two if there is any sensation of faintness. In these instances, whenever possible, blood pressure readings are recorded. Whereas some patients show a desired effect after the intake of 750 mg. in twenty-four hours administered in divided doses, others may require as much as 6 grams daily. There is no relationship between the height of the blood pressure and the dose required for a satisfactory response.

Unfortunately, it is the diastolic blood pressure which is most difficult to influence. In some instances the amount of the drug has to be increased, even after a satisfactory maintenance dose has been reached. A booster dose given parenterally once or twice daily, and gradually increased until the desired effect is obtained, seems to re-establish the effectiveness of the compound in many cases. In other instances, discontinuance of the drug for a few days seems to have a similar effect.

TYPES OF PATIENTS STUDIED

In our series of 150 patients 58 per cent suffered from idiopathic (essential) benign hypertension, 6 per cent from idiopathic (essential) malignant hypertension, and 16 per cent from atheromatous (16, 17, 18, 19), endocrine or mixed hypertension. The remaining 20 per cent were cases of chronic glomerulonephritis, bilateral polycystic kidneys, Kimmelstiel-Wilson's disease, toxemia of pregnancy with mounting systolic and diastolic blood pressure and albuminuria, and 1 case of pheochromocytoma. Only the first three groups mentioned were treated in sufficient numbers to warrant any conclusion.

THERAPEUTIC RESULTS

The effectiveness of methonium was judged by the patient's symptomatic response rather than by the blood pressure readings alone. In our series we noted relief of such symptoms as headaches, palpitation, vertigo and effort angina in over 60 per cent of the patients treated. This symptomatic relief is of particular importance, since the same patients had been previously treated by other methods without the same degree of subjective improvement. Improvement was seen in 4 of 9 cases of idiopathic (essential) malignant hypertension; in these 4 the blood pressure so far has remained under fair control.

The results obtained with methonium in a group of 11 sympathectomized patients whose symptoms returned in a more severe form in from one to five years following surgery, are particularly noteworthy. Eight of the 11, whom we have observed for about eighteen months, showed both subjective and ob-

jective improvement. All of them take the drug both orally and parenterally, and they have learned to judge the needed dose. The improvement in all cases may be attributed to rest periods for the cardiovascular system afforded by the action of the drug.

In 11 per cent of our series, the patients could not tolerate the drug or were afraid of the effects. Approximately 27 per cent did not show any improvement in spite of persistent continued trial. Four patients died:

1. Female, white, aged 36 years. She was admitted with advanced idiopathic malignant hypertension and uremia. Following a test dose of methonium an unexpected drop in blood pressure occurred from 260/160 to 100/? Shock resulted, from which she did not recover. She remained comatose, although the blood pressure reading after eight hours returned to 160/90. Death occurred after six days.

2. Female, white, aged 60 years. She was known and treated by us over a period of fifteen years for idiopathic benign hypertension. There was improvement for six weeks after inception of methonium therapy. The patient discontinued the use of all medications for eight days. Death occurred within two hours, following an acute attack of pulmonary edema, probably with left ventricular failure.

3. Male, white, aged 54 years. He was admitted in a state of uremia. Therapy reduced blood pressure but blood urea nitrogen and creatinine levels continued to rise and the patient died of uremia eight days after inception of methonium therapy.

4. Male, white, aged 34 years. He was admitted with diffuse nephritis, azotemia and marked hypertension. For three weeks following inception of therapy the patient seemed to improve; then he suddenly died in his sleep. Permission for autopsy was not granted.

None of the deaths in our series could be directly attributed to the action of methonium.

Untoward effects

The most significant ill effects following the use of methonium are:

1. Postural hypotension, relieved by assuming the supine position. Some investigators recommend the use of epinephrine or nor-epinephrine in these instances. We have not found it necessary to administer either one.

2. Constipation, usually relieved by the use of prostigmine, mineral oil or mild laxatives.

3. Urinary retention, relieved by prostigmine and catherization.

4. Nausea and hiccough, relieved by carminatives.

5. Drowsiness,—a symptom to be watched very carefully particularly in advanced renal disease.

6. Amblyopia.

These side effects disappear fairly quickly as a rule. However, amblyopia and loss of accommodation may persist for months and are disturbing to the patient. Strange as it may seem, some of these symptoms disappear in spite of the continued use of the drug.

Drowsiness and a rash may be caused by methonium bromide. When this occurs, a change to chloride or bitartrate compounds may quickly eliminate these symptoms. Vomiting as an early symptom has been reported in the English literature in 3 instances. One fatal outcome from paralytic ileus has been reported.

In our series of patients the best results were obtained in benign idiopathic (essential) hypertension. In renal hypertension, the blood pressure was lowered without affecting the course of the disease.

CONCLUSION

In our series of cases there has been no evidence of tissue or organic damage resulting from the use of methonium.

Methonium compounds appear to be the most effective drugs at our disposal, so far, for the control of hypertension.

Both patient and physician must be continually aware of the possible side effects of methonium.

The dosage of methonium must be standardized for each patient.

Addendum

Since this paper was presented, we have found that a polypharmaceutical approach to the treatment of hypertension is most satisfactory. The use of barbiturates, rutin, *Rauwolfia serpentina*, and pentapyrrolidinium bitartrate, in combinations, has afforded the best results, so far.

REFERENCES

1. Fahr, A.: Hypertension heart, *J. Med. Sci.* **175:** 453 (Apr.) 1928.
2. Golblatt, H.; Lynch, J.; Hanzal, R. F., and Summerville, W. W.: Studies on experimental hypertension; production of persistent elevation of systolic blood pressure by means of renal ischemia, *J. Exper. Med.* **59:** 347 (March) 1934.
3. Landis, E. M.: Essential hypertension, *Mod. Concepts Cardiovasc. Dis.* **12:** part II (Sept.) 1943.
4. Craig, W. M., and Brown, G. E.: Unilateral and bilateral resection of major and minor splanchnic nerves: its effect in cases of essential hypertension, *Arch. Int. Med.* **54:** 577 (Oct.) 1934.
5. Adson, A. W., and Brown, G. E.: Malignant hypertension; report of case treated by bilateral section of anterior spinal nerve roots from sixth thoracic to second lumbar inclusive, *J.A.M.A.* **102:** 1115 (Apr. 7) 1934.
6. Scurr, C. F.: Use of decamethonium iodide in anaesthesia for peroral endoscopy, *Brit. M. J.* **2:** 1311 (Dec. 9) 1950.
7. Paton, W. D. M., and Zaimis, E. J.: Clinical potentialities of certain bisquaternary salts causing neuromuscular and ganglionic block, *Nature* London **162:** 810, (Nov. 20) 1948.
8. Kay, A. W., and Smith, A. N.: Effect of oral hexamethonium salts on gastric secretion, *Brit. M. J.* **2:** 807 (Oct. 7) 1950.
9. Wolffe, J. B.: Therapy of tissue extract (Desympatone), *Tr. Am. Therap. Soc.* 1931, p. 31.
10. Arnold, P.; Goetz, R. H., and Rosenheim, M. L.: Effect of pentamethonium on the peripheral circulation, *Lancet* **2:** 408 (Sept. 3) 1949.
11. Wolffe, J. B.: Methonium compounds in the treatment of essential benign and malignant hypertension before and after sympathectomy, (Proc. Am. Soc. Study Arteriosclerosis) *Circulation* **4:** 474 (Sept.) 1951.
12. Grimson, K. S.; Orgain, E. S.; Rowe, C. R., and Sieber, H. A.: Caution with regard to use of hexamethonium and "apresoline," *J.A.M.A.* **149:** 215 (May 17) 1952.
13. Wolffe, J. B., and Danish, A. W.: Subconjunctival hemorrhage during administration of rutin; report of 2 cases, *J.A.M.A.* **134:** 692 (June 21) 1947.
14. Plungian, M. B.; Munch, J. C., and Wolffe, J. B.: The effect of rutin on coagulation time of rat blood, *Pharmacol. & Exper. Therap.* **93:** 383, (Aug.) 1948.
15. Wolffe, J. B., and Plungian, M. B.: The effect of parenteral rutin-methylglucamine (a new organic compound) in the treatment of diabetic and hypertensive retinopathy, (to be published).
16. Wolffe, J. B.: Atheromatosis to be distinguished from arteriosclerosis, *Mil. Surgeon* **97:** 92 (Aug.) 1945.

17. WOLFFE, J. B.: Arteriosclerosis at present—A melting pot of arteriopathies, *Geriatrics* **2**: 296 (Sept.–Oct.) 1947.
18. WOLFFE, J. B.; HYMAN, A. S.; PLUNGIAN, M. B.; DALE, A. D.; MCGINNIS, G. E., AND WALKOW, M. B.: Studies in experimental atheromatosis. Atheromatosis and hepatoatherosis in geese: possible reversibility and clinical implications, *Gerontology* **7**: 13 (Jan.) 1952.
19. WOLFFE, J. B.: Atheromatous coronary artery disease, *Tr. Am. Coll. Cardiol.* **1**: 122, 1951.

MEDICAL ASPECTS OF PHYSICAL TREATMENT IN GERIATRICS

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INTRODUCTION

In a wide variety of medical problems presented by the older patient, the practitioner must use physical agents as part of an integrated comprehensive therapeutic regimen. The prevention of prolonged disability following illness or injury is of prime importance in this ever increasing group of patients, yet the basic physiologic changes during aging tend to favor or accentuate the development of disability. Among these progressive idiopathic changes (1) may be included the following:

1. Decrease in tissue elasticity.
2. Retarded capacity for cellular division and growth.
3. Delayed tissue repair.
4. Decreased speed, strength, endurance and coordination of neuromuscular reactions.
5. Decreased strength of skeletal muscle.
6. Progressive degeneration and atrophy of the nervous system; impaired vision, attention, memory and mental endurance.

Trauma or infections in the aging patient will invariably speed up the progress of many of these changes, so that a vicious cycle develops. The time required for repair and convalescence increases and this, in turn, with its attendant "immobilization" further tends to produce prolonged disability, which results in more delayed convalescence.

PHYSICAL TREATMENT

Thus, the areas in which Physical Medicine and Rehabilitation are helpful in managing the medical problems of the geriatric patient become clear. The agents and techniques of this field are indicated in the control of any factors which tend to produce prolonged disability or delayed convalescence. More specifically, the indications include:

1. The relief of pain.
2. The improvement of skeletal muscle function in terms of strength, endurance and coordination.
3. The maintenance of range of motion in the joints (prevention and correction of deformity).
4. The improvement of arterial blood flow to any given part.
5. The maintenance of adequate venous and lymphatic return (prevention or correction of peripheral edema).

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6. The enhancing of neuromuscular reactions.
7. The improvement of gait, with or without appliances.
8. The providing of motivation.
9. Insuring a maximal level of daily activity performance.
10. Coordinating the medical, social, psychologic and vocational facets of rehabilitation to achieve maximal self-sufficiency and to insure consideration of the patient as a "total human being."

It is not possible, within the scope of this paper, to discuss the details of techniques of physical treatment. Rather, an attempt will be made to present some practical considerations of therapy with reference to some of the more commonly encountered medical conditions of importance.

CLINICAL MEDICAL PROBLEMS

Hypertrophic arthritis or, perhaps more accurately, degenerative joint disease is invariably present in the later decades. It may be localized or generalized. There is poor correlation between the extent or severity of involvement and the amount of symptomatology noted by the individual. It is imperative that the physician explain the nature of this disease process to the patient, stressing its irreversible and usually progressive nature. He should instill a feeling of optimistic realism in the patient's approach to the problem of handling a usually benign and non-incapacitating disorder. The therapeutic regimen must be comprehensive and balanced. Physical treatment is a valuable part of this regimen but, because of the chronic nature of the disorder, every effort should be made to teach the patient or some responsible member of his family how to carry out this therapy at home. This is also important from the standpoint of economics and convenience.

Heat is indicated for the relief of pain. In most instances superficial techniques are adequate. Hot packs, paraffin, infrared radiation, luminous heat and warm tubs are included in this category. Short wave diathermy or microwaves are occasionally necessary for the production of deep heat. Their use should be restricted to the office or hospital; hence they are not practical in a long-range treatment program. In many patients, the presence of arteriosclerotic obliterative vascular disease precludes the use of any intense heat locally and permits the use of only the mildest heat directly over the peripheral joints, as will be noted later. The average treatment session should maintain the heating agent for about twenty to thirty minutes and this should be repeated as frequently during the day as is necessary.

As a rule, a light sedative massage following the heat will add to the patient's comfort. An active exercise program attempting to maintain mobility of the involved joints should round out the treatment session. Degenerative changes in certain locations may require additional techniques For example, in hypertrophic arthritis of the cervical spine with narrowing of the intervertebral foramina, a root syndrome may exist. Traction, either ambulatory or fixed, may be necessary in addition to the aforementioned approach. Relief is often dramatic. Involvement of the vertebral column may require, in addition, postural training and attention to the details of weight-bearing and gait.

Rheumatoid arthritis in the aged patient is a more difficult problem to handle. What has been said about the use of physical agents in the hypertrophic variety applies here. However, deformity frequently complicates the picture and more assistive and passive exercises are needed as corrective procedures. Rest is apt to be more important, as is avoidance of joint trauma. As a rule, a home program of heat, massage and exercise will be beneficial.

Fibrositis, a chronic inflammatory process involving white connective tissue, is perhaps the commonest form of rheumatic disease and is most frequently responsible for pain and stiffness, although involvement is all periarticular. It may be primary or secondary to arthritis, infectious diseases or trauma. The history is characteristic, *i.e.*, "jelling" of muscle action, which is worse on arising, and following stress, fatigue or emotional disturbances. The physical examination, if it includes adequate deep palpation of soft tissue, will reveal discrete, hard, tender and movable "nodules" or "bands" in the trapezii, rhomboids, pectorals, spinal erecti and intercostal muscle groups. The treatment of choice is superficial heat followed by a firm "fibrositic" massage over the involved areas, and mobilizing exercises. Occasionally, procaine or ethyl chloride spray techniques for "trigger areas" are of additional benefit.

Another extremely common and important group of diseases in geriatrics involve the *cardiovascular system*. Pain, trophic changes and decreased functional capacity of the lower extremities are frequent findings among geriatric patients and are produced by *peripheral vascular disease*, especially progressive obliterative arterial disease. Physical agents are of value in promoting the development of increased arterial blood flow to the extremities and in encouraging venous and lymphatic return. Heating agents should be applied carefully to prevent further embarrassment of the circulation. Mild luminous heat, infrared radiation or moist heat are most commonly used. It may be necessary, in some instances, to apply heat in an area proximal to the extremity, for example, diathermy to the lumbo-sacral area, with towel insulation of the legs. Heat applied to the upper extremities may result in an adequate reflex increase in blood flow to the lower limbs. *Hypotension* may produce troublesome fatigue. If the process is "primary" and not associated with severe cardiac involvement, a program of mild and progressive conditioning and heliotherapy is indicated. The *hypertensive* patient is usually overimpressed with the possibilities of impending disaster, and excessive inactivity is prescribed. Physical measures which promote relaxation and maintain a good state of conditioning through mild therapeutic exercise are indicated. A similar problem arises with regard to the convalescent activity status of the patient who has had a coronary occlusion. In the aged patient vocational retraining is not a factor; however, judicious use of the edict "take it easy" is suggested. Activity should be correlated with objective evidences of tolerance and the psychologic state of each patient.

The geriatric *pulmonary invalid* deserves some attention (2). Many patients with chronic bronchitis, bronchiectasis and asthma, present functional limitation of ventilation with secondary organic phenomena such as a "frozen" rib cage, muscle atrophy and poor mechanical body alignment or posture. Accessory muscles are used as substitutes for normal respiratory muscle action and a

good deal of fear and insecurity complicates the problem. Instructional sessions in relaxation, breathing re-education and postural training will assist in mobilizing the rib cage and reducing disability.

The *hemiplegic* patient is a serious problem (3). From the medical standpoint it is unwise to confine such patients to prolonged absolute bed rest. Unless cardiac failure or severe hypertension complicate the course, it is perfectly safe to begin physical treatment early. Such treatment, at the outset, attempts to prevent deformity and to assist in early re-education of muscle groups. Proper neutral positioning in bed and daily passive movement of the involved extremities through the normal range of motion are important in this regard. When spasticity supervenes, return of function will become evident and voluntary retraining should become more intensive. As soon as the quadriceps can work against gravity, standing and balancing should be added to the program. Early attempts at gait training will normally follow. Since the upper extremity shows slower return of function, it is important to make the patient ambulatory as soon as possible. Functional retraining of the hands and of speech can then proceed on an out-patient basis over a longer period of time. If deformity is not prevented the chances of functional retraining are seriously impaired. Most hemiplegic patients can at least be taught to ambulate, to perform self care, and to take part in many daily activities.

SUMMARY AND CONCLUSIONS

Basic physiologic changes in aging which influence the development of deformity are presented.

Indications are given for the use of physical medicine and rehabilitation in medical geriatric problems.

Some of the more important clinical medical problems of the aged patient are discussed in the light of the potential usefulness of physical measures in their management.

It is stressed that physical treatment is of value in assisting the physician in preventing prolonged disability and convalescence in the older medical patient.

REFERENCES

1. Carlson, A. J.: *in* Stieglitz, E. J.: Geriatric Medicine. Philadelphia, W. B. Saunders Co., 1944, chap. 2, pp. 51–71.
2. Dinken, H.: *in* Krusen, F. H.: Physical Medicine and Rehabilitation for the Clinician. Philadelphia, W. B. Saunders Co., 1951, pp. 221–227.
3. Dinken, H.: Physical treatment of the hemiplegic patient in general practice, *J.A.M.A.* **139:** 1255–1257 (Apr. 30) 1949.

PATTERNS IN AGING

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In the "Old Man's Guide" John Hill[1] in 1771 wrote that "Old men's diseases are hard to cure, but are easy to prevent. It must be *a good natural fabric* which has preserved itself so long; and the same strength may keep it much longer well, *under good regulation*"[2] (1, 2). These are ingenious observations of particular medical aspects in the elderly. One defines the constitutional biologic assets contributory to health in the later years of life, and the attributes of fortunate genetic design. The other is an expression of the need for specific basic care. Even in the older age group there are therapeutic potentialities for pathologic states present in, and associated with, but not necessarily secondary to, aging.

Good fabric and sound regulation are mutually contributory to healthy longevity. Their potential benefits are many: a longer and healthier life, a longer reproductive period, a greater span of productive maturity, and improved control over those degenerative diatheses which statistically parallel the increased number of years. (It also suggests a longer period of senility).[3]

Society is being forced to the rationalization that it is better to treat a vascular limitation at 70 years than to have recorded a decease from infection in infancy or childhood. There is no volition in this rhetorical expression of a biologic occurrence. The clinician managing diseases of older patients does not deal in theories of prolonging life; he is trained more particularly in physiologic principles of gerontologic medicine. As anachronistic critique of this modern view is found in the quarrelsome Hill:

"Lord Bacon laments justly that physicians have applied themselves solely to the cure of diseases, neglecting the prolongation of human life."

Most of the gains thus far made in the changing patterns of population have been accomplished at the expense of gross infections, and only a small percentage against other, usually less dramatic, limiting diseases. As infections are restrained by specific agents, maximal benefit is yet to be obtained from measures which

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[1] Zeman said of him "Lest any time be wasted on this worthy . . . we quote the summation of Hill's character to be found in the Dictionary of National Biography: 'A versatile man of unscrupulous character, with considerable abilities, great perseverance, and unlimited impudence'."

Garrick added: "For physic and farces, his equal there scarce is,
His farces are physic, his physic a farce is."

[2] Italicized by author.

[3] Senility is somatic, physiologic and psychologic inadequacy in a social complex, due to aging.

might limit the chronic syndromes due to changed metabolic processes. Possibly the immunologic characteristics of the higher-aged body react to *subclinical* infections operative over longer periods to produce modified disease patterns. Duran-Reynals has studied metaplastic and neoplastic conditions from this viewpoint (3). For the most part, such concepts are still in the early stages.

Geriatrics therefore is a discipline which had to happen—the inevitable invention of man who, in improving himself (and his stock), created the problems of his own and his family's longer life. Widening control of pathologic conditions must eventuate in longer life. These additional years have clinical implications to be met in some positive manner that is more than a medical afterthought. The major successes in the treatment of diseases of the younger age group have resulted in more years of living. An adjustment in society for the increasing numbers and greater proportions of older persons is overdue. Effective campaigns against single disease entities have contributed to longer life with more survivors but without an adequate plan of what to do with the end results. Medicine cannot thrive in this social void.

Too many reports in geriatrics are collections of data bounded by a dateline or birthdate, without further characterization. Principles underlying the patterns of aging have been obscured by unwarranted adaptations from other phases of the life span. This tendency is a distortion. It has encouraged a sense of resignation to the sweep of those pathologic processes attaining an acme in the older system. It has tended to obscure those accumulated effective mechanisms of which the body in later life is a familiar banner in itself. This vital mechanism is a major accomplishment, a temporary victory over complex pressures tending to terminate the cycle of living. Failing to do so over a period of a century or more in some instances, these forces have molded the biologic structure into a framework identified structurally as old age, of which the components may be isolated as particular responses to separate stimuli. In this sense, aging is the summated somatic expression of all reactive states in time. Its physiologic normal differs from the pathologic abnormal only in degree and characteristics proportional to the time, duration, and nature of the stressing agency. The end stage is the resultant of many vectors recognized clinically as pathologic syndromes.

It is almost impossible to designate at what age a gerontologic study should begin. There are personal deviations from the average ranges. In some diseases there are obvious similarities between calendar-old and calendar-young patients. Some illnesses seem to erase the differences in years and place the familiar mask of age on both young and old. When the primary disease is halted or reversed this mask may be lifted selectively, or it may persist. Custom has drawn the line fairly accurately; the years from 40 to 60 contain the major vital determinants. Generally in sixty years of living the dominant influences of the total life span have stratified so that the basis of eventual finiteness is increasingly apparent. Medical convention accepts six completed decades of living as a date of geriatric expression, and this approximate time fits the current scheme of social arrangements. With these several qualifications, age for an individual is a matter of physiologic resources.

Regardless of this clinical approximation, it is questionable whether it is always possible to distinguish between states of being old pathologically and states of seeming old naturally. Frequently these two must be almost inseparable, which voids a clear-cut delineation. It then must be concluded that what is called old age is but a familiar syndrome which might be modified by a cogent interference with those processes giving rise to the vestments of age.

Furthermore, it is a presumption to designate certain stages of life as irreversible old age. Malfunction and dysfunction can be, and are operative in the higher brackets. What age is the requirement for the definition of premature aging? Since progeric agencies in younger years can be corrected by proper intervention, it is not incongruous to suggest that there is a progeria, even in the accepted range for old age, for which specific measures may be discovered. This has been true of many disease states, once considered irremediable. It is resolved into a question of the discovery of, and response to, effective treatment. These considerations stress the relativity of health and disease in all ages and extend the scope of potential vital values.

Limitations of many types are compatible with longer life, and a multiplicity of pathologic phenomena is consistent with lengthy unimpaired living. A list of diagnoses with overtones of abnormality characterizes most older bodies. Such tabulations are not necessary restraints on successful living. A man is as old as his physiologic lability; he stands off the onslaught of pathologic time as long as he has an equity in homeostasis. When there is a failure to respond with the minimal essentials necessary to maintain balance, the result which might have been a resumption of successful living, becomes either a limitation of that state or a terminus to life.

During the middle years of the twentieth century there have been marked modifications in morbidity and mortality tables based on specific anti-infection measures in addition to the general principles of sound hygiene. The antibiotics have made giants out of physicians. Average life expectancy at birth exceeds the traditional 70 years. With the erasure of this ancient and unscientific limitation it is reasonable to wonder what will be the ultimate length of life under ideal conditions. Rolleston referred to the ideal physiologic duration of life as 100 years. He quoted Flourens as concluding that "the natural duration of life" was the same length of time. Buffon, he states, came to a similar conclusion by multiplying the age of puberty by seven (4). By these yardsticks, modern man in the middle of the twentieth century has a life span which is appreciably short of its inherency.

The question of mere existence is not the point. Organic continuity derives little satisfaction from just having survived, as in the couplet[4]:

> "For fourscore years this life Cleora led,
> In the morn she rose, and at night she went to bed."

The goal is a useful and vital span with a minimum of vegetative living. To this end many factors of security must be pointed. Current social forms are still

[4] Quoted from memory.

somewhat immature in the management of such an adult society. Security in longer living must be combined with all elements which constitute sociologic adequacy.

The object of medicine is to increase the length of healthy life; the object of society in part must be to establish the proper environment to justify this living span. There can be no artificial division between them, since they are complementary halves of organic living. Long living and cultural security connote degrees of responsibility which exceed those which have been accepted as sufficient. Physicians cannot deny their role, since they have promoted both aspects and to some respect benefited from them. The fields of social rehabilitation cannot be evaded by dogmatic limitation to the mechanics of diagnosis and treatment. Medicine has a major place in all of aging, its promotion, its guidance, and its ultimate welfare.

The physician of the current "physiologic school" in clinical medicine faces a new set of social forces. These are modifying his formalized attitudes and his clinically conditioned categories. Although the environment for survival has improved, there has not been a notable increase in the inborn potential for living. Measures which modified morbidity and mortality in younger decades are reflected in later years. The prophylaxis of vascular and neoplastic conditions in middle and late maturity is taking a position comparable to the past's attitudes toward epidemic infections.

Grave questions are involved in the facts that more people are living longer, that those born now have a lengthening average span, and that those surviving from a former era benefit from current medico-social advances.

In longer living there might be an extension of the reproductive phase of existence, an expansion of the middle age of man, and an elongation of the productivity of the defervescent years. These three would be ideal. Will such symmetrical improvements in well-being extend into a longer period of relatively healthy or sick senescence? This is a good deal less than ideal. Finally, will the increasing desire to survive ever accept *pari passu* the biologic reality of not living?

Will there be a constantly increasing number of vegetative aging who require increased allocations from the world's economy, including more than proportional fractions of the reserves of medical and nursing resources?

Who shall determine comparative values of life, of what Sherrington called "a substance from the stars"? Undoubtedly there are planes of utility in which *being* is considered equal to *doing*, depending on the premise of the comparison. Even Plato found this difficult, although he did not avoid showing the trend of his harsh conclusions. The major productive members of society are a minority whose talents and energies, regardless of their individual age, add to material well-being for noncontributors. The latter benefit by longer survival. Their usefulness then is questioned as if there had been some change in their status. In this sense the measure of the right to live cannot be judged by usefulness. If it were, the world's population and the age of its populace would be shrinking.

As the likelihood of independent healthy years for a majority of those being born increases, there must be some preparation for the possbility of a lengthened

period of dependent senility. A longer functional maturity must be balanced against a longer period of totally impaired survival. The problems of the former are just beginning to get recognition—housing equal to pension incomes; avocations and recreations within physical and economic abilities; equal distribution of proper nutrition; adequate hygiene and medical care at home, in the clinic, and in hospitals; family protection against catastrophic situations; increased mobility in living in the retired years; forced, selective, or gradual retirement by age, by industry, down-grading, union contract, custom, or other policy; social acceptance of higher age's values; and personal re-evaluations.

With regard to the problems of the senile group, different progress has been made—custodial homes, private, public, endowed; foster homes for older persons; homes for the incurable; chronic disease hospitals or wards; half-way houses; nursing and boarding homes of varying standards; hotels for the aged; and home-care programs. Since the obvious needs of these senile persons are greater, they are getting more care. The major portion of the lives of today's older persons preceded many of the benefits of modern medicine which extend able and disabled survival.

The physician of the future may find more of his time and energies devoted to elderly persons who are irremediably ill, but who in spite of their pathologic straits still yearn for more days and months and years of living. Overwhelmed with their aging and sicknesses, many elderly lie stretched out and try to pour more life into barely existing bodies. Possibly methods will be devised to lengthen and strengthen selectively the better portions of life, ending in a period of senility which will be short and terminal. This does not seem likely. On the island of Nantucket in 1952 there were 66 deaths recorded (some of these did not actually occur on the island).[5] The average age at death was 68 years. The distribution was:

0–10	11–20	21–30	31–40	41–50	51–60	61–70	71–80	81–90	91–100
0	1	0	2	4	11	15	17	13	4

Only 10 per cent of these deaths occurred in people under 50 years of age and half occurred in people in the senescent years. Home-nursing services make more than half of their calls on older persons with long-term ailments, although these constitute but 8 per cent of the population. With the anticipated demographic changes, this type of service promises to get much greater.

At some point the physician may be forced to modify his traditional role with the claim that he can no longer heal, can no longer maintain physiologic balance, must use his efforts more fruitfully, and leave the remaining elements of feeble vitality to the natural sweep of terminating pathologic processes.

A 72-year-old man is hospitalized with a transecting spinal cord lesion due to a metastatic prostatic malignancy. With orchiectomy and estrogens the neoplastic process is halted temporarily. His mood, desire to live, and attitudes are vivid, enthusiastic, and courageous. These are present despite the fact that he cannot walk, is incontinent, and has associated difficulties. He says that he will walk, and in his goal there is ascendant dignity. The resources of his family are

[5] Taken from *The Inquirer and Mirror*, Nantucket, Mass. **132** (#33): 2 (Feb. 14) 1953.

turned wholly to his care and his rehabilitation. Each improvement in function is heartening. His drive to live normally has become the reason in itself for living.

A 77-year-old man has the following: diabetes mellitus, hypertension, extensive sclerotic disease involving coronary vessels, cardiac valves, and myocardium, decubitus angina, recurrent decompensation, diabetic neuropathic changes, *marche à petits pas* due to encephalomalacia, and loss of urinary control secondary to extensive resection of a large benign prostate. His hold on survival is tenacious, unyielding, and unpleasant. Every measure is used for his survival and for complying with his endless demands. At very best, only a temporary foothold on life is available to him. His drive just to live somehow, is an end in itself.

Today medical skill can maintain some balance far into hopeless pathologic conditions. The commonest example is the older patient with irreversible insufficiency. He may manifest mechanical and chemical imbalances of such depths as to seem to defy rescue. There are available drugs of such efficacy, electrolyte control of such delicacy, vascular support of such precision, as to reclaim the patient from almost impossible states. Physicians will be increasingly confronted with the problem of harmonizing the patterns of biologic aging with both the social scheme and intrinsic individual rights.

REFERENCES

1. Zeman, F. D.: Life's later years, *J. Mt. Sinai Hosp.* **12:** 939 (Nov.–Dec.) 1945.
2. Hill, John: The Old Man's Guide to Health and Longer Life, ed. 6. London, E. and C. Dilly, 1771.
3. Duran-Reynals, F.: Age and infection; a review, *J. Gerontol.* **1:** 358, 1946.
4. Rolleston, H. D.: Some Medical Aspects of Old Age. London, MacMillan & Co., 1922, p. 17.

SOME ASPECTS OF THE GASTRIC CANCER PROBLEM

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Carcinoma arising in the stomach is one of the most frequent forms of cancer in the human body and, as with other forms of malignant disease, is much more frequent in the older age group. The Bureau of Vital Statistics of the United States in 1948 reported total deaths from cancer and other malignant tumors to be 197,042, of which 13.3 per cent (26,215) arose in the stomach (Table 1). The death rate per 100,000 for all cases of cancer in 1948 was 134.9 and in 1950 was 139.6, a slight increase in all types of malignant disease.

The magnitude of the problem is reflected in the fact that from 30,000 to 35,000 persons in the United States die each year from cancer of the stomach. We can estimate that approximately three or four times that number are alive with the unrecognized disease or still living with diagnosed cancer, either inoperable or successfully resected. Thus, its recognition is a considerable problem. The over-all incidence of carcinoma of the stomach in individuals from 1 to 74 years of age is 11 per 100,000, but in patients between the ages of 45 and 74 years the incidence is approximately five times as great. It should be noted in this regard that 95 per cent of malignant tumors of the stomach occur in patients more than 45 years of age and that 74 per cent occur in patients 60 years of age or older (Table 2).

Carcinoma of the stomach, by virtue of its common incidence, its vague clinical manifestations in the early course of the disease and its tendency to spread and to metastasize rapidly, presents a real problem to the internist, the general practitioner and the surgeon alike. The ultimate results of surgical treatment, as it has been practiced in the past, have been distressingly poor. The limited salvage resulting from subtotal gastric resection has led to an increasing interest in a more aggressive approach to treatment. In general, these considerations have been directed toward 1) earlier diagnosis of the disease, and 2) the possibilities of extending the scope of surgery.

The past inadequacy of clinical detection and surgical treatment of cancer of the stomach is epitomized by the observation that of 100 consecutive individuals observed in the clinic with this disease, 26 were obviously inoperable, 74 were operated on, and of the number having exploratory operation, 38 were treated by a partial or a total gastrectomy. Of those treated by resection, in only a small percentage did the condition of the tumor appear grossly favorable, and only about 1 in 4 (26.7 per cent) of those treated by subtotal gastric resection survived five years.

A recent observation by Ochsner and Blalock (1) indicated that in a series of 210 patients with gastric carcinoma observed at the Ochsner Clinic in the ten-year period from January 1942 to January 1952, 12.8 per cent had such advanced lesions when first seen that they were considered inoperable; 87.2 per cent were suitable for operation. Seven per cent refused surgery or were operated

TABLE 1

Deaths from Cancer and Other Malignant Tumors

(Bureau of Vital Statistics of United States—1948)

	Male	Female	Total
All types of malignant tumors.....	97,944	99,098	197,042
Carcinoma of stomach.....	15,321	9,894	26,215 (13.3%)

TABLE 2

Deaths from Cancer of Stomach—Ages of Patients

(Bureau of Vital Statistics of United States—1948)

Age (Years)	Total Number of Deaths
1–19	20
20–24	26
25–29	72
30–34	163
35–39	319
40–44	592
45–49	1068
50–54	1852
55–59	2774
60–64	3570
65–69	4222
70–74	4335
75–79	3692
80–84	2282
85–89	963
90–100	267
Over 45	95.4%
Over 60	74.0%

on elsewhere; 80 per cent of the entire group had exploratory operations, 33.3 per cent were treated by resection, 29 per cent survived resection and 9.9 per cent were alive at the end of five years. These figures, which are fairly characteristic of the current status of the surgical management of carcinoma of the stomach, reflect a profound deficiency in the early clinical detection of carcinoma of the stomach and the inadequacy of past attempts at surgical control of the disease. Such statistics indicate a need for a more aggressive approach to the early diagnosis of carcinoma of the stomach and the urgency of attempting to increase the scope of surgical maneuvers in the hope of more successfully encompassing the extent of the cancer at the time of the initial resection.

CONSIDERATION OF FACTORS CONTRIBUTING TO THE POOR RESULTS OF THE CURRENT THERAPY FOR CANCER OF THE STOMACH

The fundamental inherent weakness in the methods for early clinical detection of cancer of the stomach is related to the fact that the disease may be asymp-

tomatic until it has already spread to adjacent structures or has metastasized. Early symptoms, which may appear only after the disease is surgically incurable, are vague and nonspecific. Early clinical manifestations are such as to invite delay on the part of the patient in seeking medical attention. Equally discouraging is the fact that patients with vague early symptoms of carcinoma of the stomach who seek medical care are frequently treated on a symptomatic basis, without adequate study. In a recent spot analysis of 300 cases at the clinic we found that in approximately one third (93 cases) the physician was responsible for the delay in surgical treatment of this condition. One third of the patients had been treated for periods ranging from three months to two years, without roentgenologic examination. In a little more than one third (118 cases) the patient was responsible for delay in treatment. Of the entire group of 300 patients only 89 received surgical treatment within three months of the onset of their symptoms.

SUGGESTIONS FOR THE EARLIER DETECTION OF CARCINOMA OF THE STOMACH

Many methods have been suggested in the hope of improving the early diagnosis of cancer of the stomach. It was hoped that by mass roentgen surveys, asymptomatic cancer or its precursors would be detected in a significant percentage of cases, but several efforts in this regard have proved discouraging. For instance, Roach and his associates (2) in a recent mass roentgen survey found asymptomatic cancer or its precursor in only 1 of every 476 examinations. Although it is true, as pointed out by Ochsner, that roentgen examination gives reliable results in approximately 95 per cent of cases of gastric carcinoma now seen clinically, this figure is more a reflection of the hopelessness of the lesion from the therapeutic standpoint at the time of study, than an indication of the sensitivity of roentgenologic tests in this disease. Ochsner expressed the hope that in the not too distant future it would be possible to diagnose most cases of gastric carcinoma before the growth is large enough to be visualized roentgenographically. This statement is perhaps more hopeful than prophetic. Improvement in diagnostic techniques, such as cytologic study of desquamative epithelium by the abrasive balloon maneuver (Panico, Papanicolaou and Cooper (3)), may yet prove of value. Gastroscopic examination, although by no means infallible, frequently gives valuable information as to the presence of carcinoma, and suggestive evidence for or against its presence in ulcerating lesions that are demonstrable by roentgenologic study. More promising methods of screening the population in the hope of detecting early cancer may concern the recognition of some of the common precursors of cancer. In our experience at the Lahey Clinic for instance, 64 per cent of patients with all types of tumors of the stomach (97 per cent of which were cancerous) were males, and in 90 per cent of these cases the patients were past the age of 45 years. We also know from our own experience and from that recorded in the literature that the incidence of cancer of the stomach in patients with pernicious anemia is two to three times greater than it is in the average cross-section of the population. The presence of hypochlorhydria or achlorhydria should make the physician highly suspicious of a malignant lesion of the stomach. Polyps arising from the gastric mucosa, like other types of ade-

nomatous polyps of the gastrointestinal tract, are often premalignant. Cromer, Comfort and Butt (4), in a series of 195 cases of gastric polyps, found that 35 (18 per cent) were malignant. Opinion is greatly divided as to the significance of chronic gastritis as a precursor of cancer, but there is enough evidence regarding this relationship to warrant careful consideration and to make the physician search for carcinoma. This is particularly true of patients with prepyloric or antral gastritis. These individuals should be examined carefully and repeatedly for evidence of malignant degeneration and when roentgenologic abnormalities persist, they should have exploratory operations in order not to overlook an early cancer.

One of the most controversial aspects of the consideration of the precursors of cancer of the stomach is concerned with the relationship of gastric ulcer to gastric cancer. Allen (5), in an analysis of cases observed by him before 1939, found that 14 per cent of the patients who were operated on for benign ulcer had cancer of the stomach. A survey (6) of his more recent material indicated that the incidence of error in diagnosis was about 10 per cent. Engel (7) found that it was impossible to distinguish between benign and malignant ulcers of the stomach by roentgenologic technique in 34 per cent of his cases. In a study of 600 cases of gastric ulcer by Smith and Jordan (8) at the Lahey Clinic, 270 patients were operated on because the ulcer did not heal, because it recurred, or because it was initially suspected of being malignant. In 59 of the 270 cases, or approximately 22 per cent, the ulcer proved to be malignant. There are many opinions regarding the causal relationship between chronic gastric ulcer and gastric cancer, ranging from Allen's conviction that benign gastric ulcers do not become malignant, to the contention of Wilson and MacCarty (9) in 1909 that in a series of 153 cases of proved gastric carcinoma, 71 per cent presented evidence that the cancer originated in a pre-existing ulcer. Stout (10) observed that in 13.4 per cent of 82 cases in which gastrectomy was performed for cancer, there was evidence that the cancer had developed on the margin of the pre-existing ulcer. Although it is impossible to say precisely what the relationship is between benign gastric ulcer and gastric cancer, it is our opinion that a chronic gastric ulcer rarely, if ever, undergoes malignant degeneration. Shields Warren (11) tells us that in only 4 instances in his laboratory was there histologic evidence that carcinoma arose from a benign ulcer. The question, then, is one of initial recognition rather than one of detecting a transformation. The practical aspect of this problem is, in the case of ulcer, to decide which is benign and which is malignant. Although ulcers on the greater curvature are frequently malignant and those in the prepyloric area are more commonly so, the only practical criterion for distinguishing a benign from a malignant gastric ulcer is failure of the ulcer to heal rapidly and to remain healed.

There is a growing conviction, among surgeons particularly, that all gastric ulcers should be treated surgically. This conviction is predicated upon the observation that the incidence of cancer in gastric ulceration is high, and that the differentiation between a benign and malignant ulcer of the stomach cannot be made with certainty on the basis of history, roentgenologic or gastroscopic find-

ings; and perhaps also upon the misconception that benign gastric ulcers frequently undergo malignant degeneration. If, however, as we believe, a gastric ulcer rarely, if ever, undergoes malignant transformation, this problem can be reduced to a consideration of distinguishing between benign and malignant ulcer of the stomach. Obviously, the surgeon should operate on all patients with ulcers on the greater curvature, and on all those with recurrent gastric ulcers, prepyloric ulcers and ulcerations associated with pernicious anemia. In determining the incidence of gastric malignancy in operative specimens of so-called gastric ulcers, it should be borne in mind that this incidence does not necessarily represent diagnostic errors, since most of these patients underwent operation precisely because of the impossibility of excluding carcinoma. Indeed, in a series of 411 patients undergoing gastric resection for gastric ulcer, the indication for operation in 195 was the inability to rule out the presence of carcinoma. The other aspect of this problem is emphasized by the fact that a definite diagnosis of carcinomatous ulcer was made preoperatively in 24 of a group of 346 patients who had pathologically proven benign gastric ulcers. Eusterman (12) has summarized the clinical characteristics which strongly suggest the presence of cancer in gastric ulcer. These include recent onset of an ulcer of large size located on the greater curvature or the prepyloric area, and a progressive clinical course; histamine-fast achlorhydria; prepyloric obstruction; persistent occult blood in the stool and an increase in the size of the lesion while the patient is under careful medical treatment. Although it is true that a malignant ulcer of the stomach may show some response to medical treatment, both in terms of symptomatic relief and diminution in size of the ulcer, such an ulcer will not heal completely under medical treatment.

DIAGNOSIS

It is obvious that earlier diagnosis of carcinoma of the stomach is essential for a significant reduction in the mortality from this disease. Earlier recognition will depend upon an awareness on the part of the physician of the frequency of carcinoma of the stomach, particularly in men more than 45 years of age, and especially in those patients 60 years of age or more. More importantly, recognition depends upon the realization that the classic symptoms and signs, such as abdominal pain, marked loss of weight, vomiting, tarry stools and the presence of a palpable epigastric mass, are usually indicative of far advanced lesions that are already beyond the hope of cure. We should cultivate an attitude of suspicion which leads us to look for cancer of the stomach before the so-called characteristic symptoms appear. We should bear in mind the significance of persistent vague indigestion associated with anorexia, mild weight loss and fatigue, as an index of possible cancer. We should insist that any individual (especially one in the older age group) who has indigestion or gastrointestinal distress which does not respond rapidly to simple dietary and medicinal management, have careful fluoroscopic and roentgenologic examination by an experienced roentgenologist in order to establish the presence or absence of such a lesion. Failure to demonstrate a gastric lesion by these methods does not relieve the physician of the

responsibility of pursuing the matter further. Repeated roentgenologic examinations at short intervals may be necessary in order to detect early cancer of the stomach. Any suspicious alterations in contour, motility or flexibility of the gastric wall, even though not diagnostic of cancer, indicate the need for gastroscopy. Finally, as has been repeatedly emphasized by one of us (S.F.M. (13)), it is necessary to perform exploratory operations on a greater number of patients, who have vague gastrointestinal symptoms and whose histories suggest cancer even though demonstrable roentgenologic indications of the disease are absent.

SURGICAL TREATMENT

Major factors in the poor results of surgical treatment of cancer of the stomach are the tendencies toward early involvement of the adjacent viscera and early metastasis. These tumors spread not only by way of the lymphatic channels, but also by blood vessel invasion and by intramural extension. The anatomic relationships of the stomach, and particularly the points of origin of the major arterial trunks to the stomach, do not permit the wide excision of perigastric lymph-bearing tissue which is possible, for instance, in resection of the large intestine.

Several authors have recently presented material which stresses the difficulty of determining at the time of the operation the extent of the carcinoma. Walters, Gray and Priestley (14) noted that in one third of their cases of cancer of the stomach, death occurred as the result of recurrence in or about the stomach. In our experience 19.6 per cent of recurrent carcinomas occurred in the remaining part of the stomach. McNeer and his associates (15), in analyzing an autopsy series of 92 patients who had gastric resection for gastric carcinoma, found evidence of recurrence of carcinoma at the gastroenterostomy site or in the wall of the stomach in 50 per cent. An additional 10 per cent showed recurrence in the duodenal stump, and 21 per cent in the perigastric nodes or in the perigastric tissue. Coller and his associates (16) carefully examined resected specimens for evidence of extension into the duodenum and found such evidence in 26.4 per cent of patients. Harvey and his associates (17) found duodenal involvement in 20 per cent of resected specimens and esophageal involvement in 37 of 270 cases. Ranson (18) made detailed studies of specimens removed from the upper and lower ends of operative specimens in 44 cases of total gastrectomy. He found evidence of extension to the esophageal line and to the proximal end of the resection in 21 patients and involvement of the duodenum in 13 patients. Because of a comparable experience, McNeer has advocated immediate frozen-section analysis of each end of the resected specimen at the time of initial operation in order to determine whether a larger amount of tissue should be removed.

In a series of 469 patients with carcinoma of the stomach observed at the clinic from 1936 to 1940 inclusive, 48.2 per cent were inoperable and 19.4 per cent had exploratory operation and biopsy only. Palliative operation was performed in 8.3 per cent and partial gastrectomy was possible in 24.1 per cent. That some progress is being made in this direction is indicated by the fact that from 1941 to 1945 inclusive, 230 patients were observed who had carcinoma of the stomach,

and of these, 13.5 per cent were inoperable. Exploratory operation was carried out in 44.3 per cent, a palliative procedure was effected in 5.2 per cent, and resection was possible in 37 per cent. Of the 85 who underwent resection, 18 had total gastrectomy, 61 partial gastrectomy and 6, a transthoracic procedure. During the period from 1946 to 1952 inclusive, 634 patients were operated on for carcinoma of the stomach. In 339, or 53.5 per cent, resection was possible (partial gastrectomy, 250 cases, and total gastrectomy, 89 cases); in 258, or 40.7 per cent, the operation was exploratory only; and in 37, or 5.8 per cent, the operation was palliative. The operative mortality in the resected group was 6.7 per cent.

The limitations of partial or subtotal resection for carcinoma of the stomach are indicated by the five-year survival rates, which vary from 20 per cent (Welch and Allen (19)) to 34.7 per cent (Pack and McNeer (20)). Our own five-year survival rate for subtotal or partial gastrectomy is 26.7 per cent. When one considers that only one third of patients originally seen with carcinoma of the stomach undergo resection, and that less than one third of this group survive five years, it is realized that the over-all survival rate of the entire group (approximately 10 per cent) is poor indeed. A realization of the poor results of partial or subtotal gastric resection in the treatment of cancer of the stomach and the difficulty the surgeon has in determining at operation the extent of malignant invasion of the stomach wall, has led to a consideration of extending the scope of resection in this disease. A study of the lymphatic drainage of the stomach indicates that partial gastrectomy in many instances is not a sufficiently radical procedure for cancer of the stomach. The necessity for removing the entire greater omentum, the spleen, a generous cuff of the duodenum, and frequently to extend the resection to include the distal portion of the esophagus, is obvious in many instances. Although it cannot now be demonstrated on a statistical basis that total gastrectomy will increase the salvage rate, since in the past total gastrectomy has been performed primarily for the removal of cancers of the stomach that would not otherwise be removable, it does seem logical that the more radical excision of these tumors along with the natural areas of lymphatic drainage would offer some hope for increasing the salvage rate. Since currently the mortality of total gastrectomy among experienced gastric surgeons compares favorably with the mortality of subtotal resection, it seems justifiable to apply the more radical procedure to a larger number of patients with malignant lesions of the stomach. At the Lahey Clinic the mortality for a group of 250 patients who had partial gastric resection (1946 to 1952 inclusive) was 6 per cent, whereas the mortality in a consecutive group of patients who had total resection (1944 to 1953) was 8.9 per cent (10 deaths in 112 total resections). Therefore the mortality rate was only slightly higher in the group who had total resection. It is also true that the operative risk in patients 60 years of age or older is not greater than that in the younger group. In a consecutive series of 100 patients whose ages ranged from 60 to 89 years, the operative mortality was 7 per cent, and the operations in this group included a large number of total gastrectomies. In the oldest patient, who was 89 years of age, a high partial gastrectomy was performed and the patient survived until the age of 95 with no recurrence of carcinoma.

Although it is not obvious that the utilization of total gastrectomy for small lesions in the distal part of the stomach will enhance the prospects for cure, it does seem probable that the more radical resection involved in total gastrectomy will give the patient who has a malignant lesion in the middle or proximal portion of the stomach a better chance for longer survival. Occasionally it is feasible and worth while to resect the stomach when a gastric cancer invades some of the adjacent viscera, such as the transverse colon or pancreas, and there is no evidence of distant metastases; for in some instances the involvement of adjacent viscera is due merely to the proximity of the tumor and does not necessarily indicate metastatic spread. All technically removable carcinomas, therefore, should be resected.

Although it is true that a patient who has had a total gastrectomy presents some real problems with regard to nutrition, it is equally true that these problems are not extreme and insurmountable. These patients can be carefully managed with frequent small feedings. An effort should be made to prevent secondary anemia and to detect primary anemia and treat it appropriately if it should occur. One reason these patients present a nutritional problem is that they frequently have persistent malignant disease. In a group of 15 patients with benign disease who had total gastrectomy at the clinic, maintenance of a normal nutritional status and blood picture has not been a problem.

SUMMARY AND CONCLUSION

Cancer of the stomach is a relatively common disease, particularly in males over 60 years of age. Without question, it is a disease of the aging population. It is difficult to diagnose early in its course because of the vagueness of its clinical manifestations. Failure to detect the disease early limits the effectiveness of surgical treatment, as indicated by the present-day low rate of operability and of survival following resection. Improvement in therapeutic results depends upon earlier clinical recognition resulting from a higher index of suspicion as to its presence, better methods of screening the susceptible population and an increasing willingness on the part of the surgeon to perform exploratory operations in suspected cases before diagnostic laboratory signs develop. The risk of operation in patients over the age of 60 is about the same (7 per cent) as that in patients of any age (6.7 per cent). Operation in the aged has not proved to be more hazardous than in younger persons, and can be performed with the same margin of safety.

REFERENCES

1. Ochsner, A., and Blalock, J.: Carcinoma of the stomach; necessity for reevaluation of therapeutic philosophy, *J.A.M.A.* **151:** 1377–1384 (Apr. 18) 1953.
2. Roach, J. F.; Sloan, R. D., and Morgan, R. H.: The detection of gastric carcinoma by photofluorographic methods. III. Findings, *Am. J. Roentgenol.* **67:** 68–75 (Jan.) 1952.
3. Panico, F. G.; Papanicolaou, G. N., and Cooper, W. A.: Abrasive balloon for exfoliation of gastric cancer cells, *J.A.M.A.* **143:** 1308–1311 (Aug. 12) 1950.

4. Cromer, H. E., Jr.; Comfort, M. W., and Butt, H. R.: Gastric acidity in cases of adenomatous gastric polyps, *J. Nat. Cancer Inst.* **10:** 497–503 (Oct.) 1949.
5. Allen, A. W., and Welch, C. E.: Gastric ulcer; significance of this diagnosis and its relationship to cancer, *Ann. Surg.* **114:** 498–509 (Oct.) 1941.
6. Allen, A. W.: Cancer of the stomach, *Surg., Gynec. & Obst.* **92:** 757–759 (June) 1951.
7. Engel, G. C.: Problems of gastric carcinoma, *J.M.A. Alabama* **20:** 149–153 (Nov.) 1950.
8. Smith, F. H., and Jordan, S. M.: Gastric ulcer: a study of 600 cases, *Gastroenterology* **11:** 575–593 (Nov.) 1948.
9. Wilson, L. B., and MacCarty, W. C.: Pathological relationships of gastric ulcer and gastric carcinoma, *Am. J. M. Sc.* **138:** 846–852, 1909.
10. Stout, A. P.: Pathology of carcinoma of stomach, *Arch. Surg.* **46:** 807–822 (June) 1943.
11. Warren, S.: Personal communication.
12. Eusterman, G. B.: Newer phases of gastroduodenal ulcer (Julius Friedenwald memorial address), *Gastroenterology* **8:** 575–585 (May) 1947.
13. Marshall, S. F.: The relation of gastric ulcer to carcinoma of the stomach, *Ann. Surg.* **137:** 891–903 (June) 1953.
14. Walters, W.; Gray, H. K., and Priestley, J. T.: Advances in the surgical treatment of cancer of the stomach, *Proc. Staff Meet., Mayo Clin.* **27:** 156–161 (Apr. 9) 1952.
15. McNeer, G.; Booher, R. J., and Bowden, L.: Resectability of recurrent gastric carcinoma, *Cancer* **3:** 43–55 (Jan.) 1950.
16. Coller, F. A.; Kay, E. B., and MacIntyre, R. S.: Regional lymphatic metastases of carcinoma of stomach, *Arch. Surg.* **43:** 748–761 (Nov.) 1941.
17. Harvey, H. D.; Titherington, J. B.; Stout, A. P., and St. John, F. B.: Gastric carcinoma; experience from 1916 to 1949 and present concepts, *Cancer* **4:** 717–725 (July) 1951.
18. Ranson, H. K.: Total gastrectomy, *Arch. Surg.* **55:** 13–30 (July) 1947.
19. Welch, C. E., and Allen, A. W.: Carcinoma of stomach, *New England J. Med.* **238:** 583–589 (Apr. 22) 1948.
20. Pack, G. T., and McNeer, G.: End results in treatment of cancer of stomach; analysis of 795 cases, *Surgery* **24:** 769–778 (Nov.) 1948.

DIAGNOSTIC AND ETIOLOGIC CONSIDERATIONS IN DEAFNESS IN OLDER PERSONS

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The full magnitude of the problem of deafness in the United States is unknown. According to the study carried out in 1935 and 1936, as part of the National Health Survey (1), a sample of approximately two and a half million persons revealed that 1 out of every 78 men and 1 out of every 85 women had a hearing handicap. Between the ages of 35 and 44 years the ratios were 1:103 men and 1:108 women. The incidence rose with each successive decade, and between the ages of 65 and 74 years hearing defects were found in 1 of every 14 men and 1 of every 18 women.

This survey, it should be noted, was made in 1935 and 1936. The ratios carry the implication of the large number of persons then in the older age brackets with partial or total impairment of hearing. It is arresting indeed to consider how those numbers have increased over the years, as the span of life has lengthened. Obviously, deafness is one of the problems which the lengthening of life has intensified.

It is fortunate that the accentuation of the problem has coincided with the new outlook on deafness which is characteristic of modern otolaryngology. The defeatist attitude with which this handicap was formerly viewed has almost completely disappeared. One reason is the increased comprehension by the otologist of the seriousness of hearing loss to the individual patient. It is not very profitable to compare the severity of one physical handicap with another, but there are a great many persons, both within and without the medical profession, who believe that deafness is an even greater affliction than blindness because, so to speak, it disconnects the individual from life. This is particularly true of persons with limited intellectual resources or with none at all. When one considers the social and financial insecurity which deafness entails, the apprehension that it brings with it, and the lack of outlet for the emotions inevitably associated with it, it is easy to understand why personality changes so often occur and why the deafened person becomes a social as well as an otologic problem.

The whole otologic outlook on hearing impairment has changed in the last decade and a half. It is no longer regarded as a misfortune which must be endured because there is nothing particular that can be done for it. The changed outlook does not, however, mean that it can be cured in most cases, or easily cured in many cases. The exact contrary is true. The significance of the change, as McLaurin (2) has recently pointed out, is that for the first time otologists are realistically facing facts and are admitting that aural rehabilitation rather than medical or surgical therapy is the solution of the problem for most deafened persons. Several years ago Morrissett (3) observed that the major otologic error

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in the past had been failure to realize that a deafened person who is not adjusted to the life he must live represents a therapeutic failure. He was speaking of nerve deafness in particular, but what he said about nerve deafness is applicable to all varieties.

It is earnestly hoped that as the new science of geriatrics develops, it will assume as one of its objectives the relief of the deafened patient. What is meant by relief is implicit in what has just been said about a realistic facing of facts. Only in occasional cases is any form of treatment likely to produce a cure, or even a significant improvement, if the impairment is total or almost total. The fenestration operation represents the most important recent advance in otology, but it could not possibly fulfil the exaggerated claims made for it in the lay press and sometimes in the medical press. Its field of usefulness is limited. This operation has nothing to offer to the nerve-deaf patient. Aural rehabilitation, on the other hand, offers a great deal to him and to all other deafened persons, and the practitioners of geriatrics would do an immense amount of good if they would help to spread that gospel. The relief of deafness in the aging person must be a cooperative endeavor, with the otologist playing his part with greater success when he has the support of all his medical confrères.

The physician who does not practice otology cannot be expected to know the details of the specialty. Equally, he cannot advise his patients properly unless he has himself some background knowledge of both diagnostic and etiologic considerations. I propose to discuss only those two considerations.

As to treatment, I shall merely say that promises of cure and relief should be carefully guarded. The patient should be told, when otologic consultation is recommended, that a great deal can be done to make life more tolerable for him. He should never be told, no matter what type of deafness he has, that his condition is curable, nor should he be promised any considerable degree of relief. Even in the cases which are most favorable from the otologic standpoint, there should be the utmost restraint in these respects. Deafness is inherently a psychic as well as a physical problem. To permit the patient to suffer a serious disappointment because therapy fails would merely increase that aspect of his defect.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The approach to the diagnosis of deafness is threefold. It requires 1) the establishment of its existence and its approximate degree; 2) its classification into one of the three general groups, namely, conductive, perceptive, or mixed; and 3) the identification of the etiologic factor (factors) responsible for the special hearing defect. In a general way, the practitioner of geriatrics, by constantly bearing the possibility in mind, can train himself to identify deafened persons. By the use of simple tests he can often determine whether the deafness is conductive or perceptive. Finally, he can often determine, at least tentatively, the etiologic factor. With this knowledge, he is in a position to advise the older patient, who, if the proper physician-patient rapport exists, will be inclined to take his advice.

The physician may find it helpful to follow the course of an otologic study:

The diagnosis of deafness, from the otologist's standpoint, begins with a careful general history, in which inquiry is made into every detail of the hearing loss. The physician who secures this sort of history will often find that he already has in his hands the clue to the etiology of the case under consideration.

A complete physical examination is the next step. The otologist should train himself to look for and recognize gross abnormalities, but the examination should be carried out by a competent internist.

The local examination includes a careful search for any abnormality of the external or middle ear or of the Eustachian tubes. The head mirror, the speculum, the electric otoscope and the pneumatic speculum should all be employed. A similarly detailed examination of the nasal, nasopharyngeal and paranasal spaces should then be carried out, since these areas are often indirectly concerned in the hearing loss.

Auditory function is appraised with voice tests, which are of no great value, and with tuning fork tests which, when properly employed, shed a great deal of light. Special tests carried out with tuning forks include, in addition to the test for upper and lower limits, the Weber test, the Schwabach test and Rinne's test.

Testing is continued with the electric audiometer, a precision instrument which produces tones of greater intensity and longer duration than tuning forks. One of its advantages is that it produces permanent records, which can be used comparatively. A single audiogram is not informative at any time; only serial testing, with a minimum of three tests, is of any value. The results of all tests should be used with discretion, which means that they must be analyzed in relation to the history and physical examination and the results of tuning-fork tests.

When the data obtained from the history, the general and local physical examinations, and the tests of auditory function have been secured and have been supplemented by such laboratory tests as are indicated, it is usually possible for the otologist to determine with a fair degree of certainty whether he is dealing with conductive, perceptive or mixed deafness. Final evaluation rests upon all available data and not upon the results of any single test.

It should also be emphasized that whereas the audiometer is an instrument for use by the specialist, the tuning fork is a precision instrument usable anywhere, at any time, and with little or no specialized training. With a tuning fork it is possible to determine the relative auditory acuity for high and low tones, the duration of air and bone conduction and, usually, the type of hearing defect. It is a great pity that its use is not made part of every physical examination.

Differential diagnosis. The characteristics of conductive deafness are as follows:

1. Hearing by air conduction is better for high than for low tones.
2. In advanced cases, loss of hearing for both high and low tones is about the same.
3. The lower tone limit may be raised, so that tones of very low frequency are not heard at all.
4. Bone conduction is normal. In the Rinne tuning fork test the response is negative, but in the Schwabach test it is prolonged.

5. The audiometric curve shows a greater loss for low tones. It is flat in advanced cases. Audiograms show bone conduction to be normal or almost normal.

The characteristics of perceptive deafness are as follows:

1. Hearing by air conduction is relatively better for low than for high tones.
2. The upper tone limit may be lowered, so that very high tones are not heard at all.
3. Bone conduction loss parallels air conduction loss and is better for higher tones.
4. In the Rinne test the response is negative and in the Schwabach test it is shortened.

The characteristics of mixed deafness are as follows:

1. There is marked loss by air conduction for all frequencies, with added or increasing loss in the high frequencies.
2. Low frequencies are heard fairly well by bone conduction, but hearing for higher frequencies is either lost or is greatly impaired.
3. The response to the Rinne test is likely to be negative with 256 or 512 dv.
4. The Weber test (256 dv) shows lateralization to the affected (or more severely affected) side.
5. When neural degeneration is superimposed on a conductive lesion, the highest tones are affected to a much greater degree than the low tones, and for all practical purposes the higher components of speech sounds are almost entirely lost.

ETIOLOGIC FACTORS

Both the physician who is conducting the general examination of the older person and the otologist who is conducting the special examination should bear in mind that much of the deafness observed is not necessarily of recent origin, even if the patient gives a clearcut history of having only recently become aware of it. Much of the deafness represents the residua of earlier disease. Unless this consideration is constantly borne in mind, the etiologic origin of many hearing impairments will remain obscure because it will not be investigated from all possible standpoints. An open mind becomes both the physician and the otologist and neither will fall into the error of assuming that because certain etiologic factors are more common than others in older persons, all hearing impairments are caused exclusively by them.

Presbycusis. Presbycusis is the most frequent cause of hearing impairment in advancing age. It is a physical loss of hearing for high-pitched tones, which comes on as the years pass—at the rate, it is said, of 1 db for each year of life. Its onset is insidious and its progress slow. Lack of attention and slowness of cerebration in older subjects increase the apparent impairment but, at that, the complaint of deafness is often greater than the results of accurate testing would indicate. Tinnitus and the recruitment phenomenon are uniformly present. In the uncomplicated case, otoscopic and nasopharyngoscopic findings are characteristically negative.

Audiometric study shows a hearing loss in all frequencies, more pronounced in the high range. In older persons the loss averages 40 db. Associated nerve

loss is the cause of the poor discrimination with respect to speech. Normally the PB (phonetically balanced) maximum score should be 90 per cent. In deafness caused by presbycusis, discrimination is only 40 to 60 per cent.

Although presbycusis is a disease of advancing age, it may begin early in life. Leisti (4), who tested by audiometric methods 451 persons between the ages of 16 and 92 with apparently normal ears, found that it may begin between the ages of 20 and 30 years and that it is more pronounced in early life in men than in women. Saxén's (5) histologic studies on 33 cases showed that central nervous system factors explained only 1 case, whereas senile atrophy of the spiral ganglion explained 13 and angiosclerotic degeneration of the inner ear as the result of sclerosis in the regional blood vessels, explained 19. His observations indicated that changes in the tissues of the middle-ear cavity and the static labyrinth were unimportant in the light of the other changes.

Otitis media. One thinks of chronic suppurative otitis media as a disease of youth, which it ordinarily is. It may, however, occur in older persons as a primary infection, or deafness may be the residual of earlier infection.

A careful history will usually make the etiology of this kind of deafness clear. It is frequently bilateral. The hearing impairment may occur promptly after the acute infection, or later, either after chronic inflammatory changes have succeeded the acute infection, or after repeated subacute infections of low virulence. In severe cases there may be actual destruction of portions of the auditory apparatus, with later formation of fibrous tissue. In chronic cases the most important pathologic change is narrowing of the Eustachian tubes, with the formation of strictures. Exuberant lymphoid tissue and coincident sinus infections increase the auditory impairment.

Otoscopic examination typically shows thickening and retraction of the drum. When the examination is made with Siegel's otoscope, fixation of portions of the tympanic membrane by adhesions may be demonstrated. In advanced cases, the location of organized strictures may be determined by Eustachian bougies. The audiometric curve in acute cases is flat, with a loss for higher tones of from 30 to 50 db. In chronic cases there is a downward slope for higher frequencies. This is one type of deafness in which the history and physical findings are so clearcut that audiometric studies are not necessary for diagnosis, even though they are essential for the record.

Otosclerosis. For the past several years otosclerosis has provided the chief interest in the management of deafness because of the dramatic results which may be achieved in selected cases by the fenestration operation. The field of this operation, as already stated, is considerably less wide than it is optimistically said to be.

The diagnosis in otosclerosis rests upon the appearance in young adults of progressive conductive deafness in spite of patent Eustachian tubes and a tympanic membrane that is completely mobile. A family history of otosclerosis is confirmatory but not essential. The confirmation provided by tuning-fork tests and audiometry is extremely important.

Histologic otosclerosis, it is estimated, occurs in about an eighth of all white

women and about a fifteenth of all white men, though in only about an eighth of the affected ears are the location and extent of the osseous lesion such that impairment of hearing becomes clinically evident. The etiology remains obscure. Otosclerosis is stimulated by pregnancy and by chronic inflammatory changes in the ear, but neither clinically nor experimentally has any relationship with other diseases been demonstrated.

For years it has been recognized that there is a psychogenic overlap in many cases, which may account for unexpected improvement after some fenestration operations. Fowler (6) has now advanced the idea that there may be emotional factors in the etiology of otosclerosis and has suggested that avoidance of emotional episodes and states, or at least a deliberate effort to shorten them, is worth trying as a method of prevention.

Syphilis. Syphilitic deafness, which is of the perceptive variety, is one of the manifestations of neurosyphilis. Curiously, very little has been written about it in the American literature although, as Tamari and Itkin (7) pointed out in their comprehensive studies, the first article on the subject appeared in the *American Journal of Otolaryngology* in 1879.

The strain of spirochete that causes syphilis may attack the eighth nerve in its entirety or in only the auditory portion. Many older patients who seek treatment for deafness have had their hearing loss for most of their lives, though it may not have become clinically evident until the tenth year, or as late as the twenty-fifth year, and their own awareness of it may have come considerably later.

This is one variety of deafness in older persons which we may confidently expect to see less of as time passes. Present methods of treating syphilis have proved remarkably effective, and there is every reason to expect that as the disease itself is brought under better and better control, the incidence of neurosyphilis, and of this particular manifestation of neurosyphilis, will become less and less frequent.

Ménière's disease. Deafness associated with Ménière's disease is of the perceptive variety. The syndrome of this disorder consists of attacks of intense vertigo, nausea and vomiting, severe equilibratory disturbances, headache, prostration, nystagmus, loud tinnitus, severe deafness and, occasionally, diplopia and loss of consciousness. Some patients also suffer from diplacusis, in which several tones are of higher pitch in one ear than in the other. Attacks occur at irregular intervals, varying from weeks to months or years. Days and weeks may be required to regain the normal status.

In typical Ménière's disease, tinnitus and deafness are likely to have been present for varying periods of time prior to the onset of definite attacks. Deafness increases with each episode and improves in the interim, but the hearing loss is progressive and, if the attacks continue, eventually becomes total. When this happens, the patient is relieved of vertigo in half or more of all cases. In the so-called symptomatic Ménière's syndrome, vertigo, nystagmus and deafness are not episodic but are constantly present.

Unless there is a very definite history of paroxysmal attacks of true whirling

vertigo of sudden onset, associated with tinnitus and with progressive perceptive deafness, Ménière's disease may be dismissed from etiologic consideration. In the absence of this special kind of history, all other symptoms and signs are inconclusive.

Acoustic trauma. The diagnosis of deafness due to acoustic trauma rests first of all upon the history of exposure to noise. The individual reaction to noise varies widely under identical circumstances of exposure. Precision diagnosis would call for a pre-exposure audiogram, but in most cases this is obviously an impossible requirement.

Auditory impairment may occur after exposure to noise, without the individual's being aware that his hearing has suffered. Studies made during the war on apparently normal soldiers showed that a large number had sustained varying degrees of cochlear damage, which for one reason or another was overlooked or was never reported to medical officers. Furthermore, a large number of men inducted with pre-existent hearing defects probably suffered enhancement of them by their wartime experiences. These men should never have been inducted, but pre-induction auditory examinations were conducted first by the spoken-voice test and later by the whisper test, both of which are notoriously inadequate. Men with disabilities directly traceable to their war experience are eligible for treatment in Veterans Administration hospitals and clinics. Those who are not aware of the origin of their hearing impairment will consult civilian otologists. These men are not yet old enough to be included in the group which is the subject of this paper, but it will not be too long before they begin to furnish a problem to the otologists of the country, who would do well to bear it in mind before it becomes apparent.

Streptomycin-induced deafness. In streptomycin-induced deafness, as in any drug-induced deafness, the diagnosis depends chiefly upon the history. The incidence of vertigo, tinnitus and eighth-nerve deafness has decreased as streptomycin preparations have improved in purity and since dihydrostreptomycin has become available. This kind of deafness is seldom observed in patients treated with small dosages and for not more than fourteen days. In tuberculosis, however, a disease in which the drug, if it is to be effective, must be given for six weeks or more, reactions referable to the eighth nerve are still observed. This type of deafness is apparently irreversible, a fact which carries its own warning against the promiscuous use of this drug.

Neoplasms. Neoplasms in the middle ear, nasopharynx, mid-brain, lateral lemniscus and thalamus may cause unilateral or bilateral deafness, depending upon their location. An infrequent cause of unilateral perceptive deafness is an acoustic neuroma originating in the sheath of the eighth nerve. Neoplasms may occur in young persons but are a far more frequent cause of deafness in older persons.

Head injury. No two head injuries are identical. The degree of resulting functional impairment therefore varies from person to person according to the site and kind of injury and its degree. Thus a transverse fracture of the petrous pyramid with destruction of the labyrinth will produce total deafness on the affected side. A longitudinal fracture will produce damage to the middle ear,

with resulting conductive deafness. In concussions, the cochlear nuclei may be damaged. After a lapse of a few weeks there is a sloping downward of the curve for the low and high frequencies by air conduction. Bone conduction is good for some frequencies and poor for others. The immediate effect of craniocerebral trauma is a profound loss of hearing for all frequencies. Deafness may also occur in fractures of the temporal bone when there is no clinical or roentgenologic evidence of the injury. The loss is of the nerve type and is most severe for high tones.

The etiologic diagnosis depends chiefly on the history, which usually includes data concerning a blow on the head. It is important to remember that although deafness may come on immediately after injury, nerve tissue degenerates slowly and the hearing loss may not be evident for six to twelve months or longer. The prognosis should always be guarded after this type of injury, which frequently has medicolegal implications.

Vascular disease. Arteriosclerotic deafness is chiefly a disease of older persons. It is usually unilateral, or is more pronounced unilaterally. Tinnitus is likely to be present in the more severely affected ear; this is one clue to the diagnosis, if the results of otoscopy and nasopharyngoscopy are negative.

The hearing loss is for all frequencies, as would be expected, because sclerosis of the internal auditory artery, which is the sole vascular supply of the labyrinth, affects the entire cochlea and diminishes its total performance. Secondary nerve degeneration follows the vascular deterioration and a greater loss for all frequencies is shown audiometrically. It is important, however, to interpret this finding, like many others, in the light of the slower cerebration of advancing age.

Fowler (8), in a report on sudden deafness in 26 personal cases, combined with an analysis of 34 other reported cases, advanced the idea that this type of deafness may be due to vascular spasm. He regarded the sludging of the blood found in the 5 cases in which it was looked for, as proof of his contention. In 23 of his 26 personal cases there was a history of emotional episodes before the onset of the sudden deafness, and such factors, as he pointed out, are known to affect the neurovascular mechanism through automatic nervous system responses. It is not possible to say at this time whether or not this theory is sound. There is no doubt, however, that only a vascular phenomenon could precipitate sudden deafness in the absence of any other evident cause.

Psychogenic deafness. Psychogenic deafness is a concept of fairly recent years, particularly the years since World War II. During the war, because it was an escape mechanism, it was of relatively frequent occurrence. It is found in the individual who is emotionally unstable, inherently neurotic in his make-up, and unable to adjust himself either to the ordinary situations of life or to situations of special difficulty. I suspect that it is a more frequent cause of deafness in aging and aged persons than we now realize, or than we shall realize until we become more cognizant of their special problems and difficulties.

Diagnosis rests upon the following points:

1. Deafness occurs suddenly, with no evident cause. It may follow a severe physical or emotional shock.
2. It is complete, or almost complete, in both ears.

3. The patient's voice is unchanged.
4. He is annoyed by loud noises.
5. Results of vestibular testing are normal.
6. There are wide variations in serial audiograms, and results of other tests are also inconsistent.
7. There is an equal drop in both air and bone conduction.
8. A hearing aid may not give the improvement which testing indicates that it should, or the improvement may be too remarkable to be accepted at its face value.
9. The response to narcosynthesis, which was brilliantly developed in the Armed Forces, is frequently very good.

The difficulty of diagnosis is that the otologist must rely, for the most part, upon the patient's controlled responses. It is significant that the audiometric and other patterns vary from test to test, although in true deafness they tend to be always of the same order. The response to narcosynthesis is at the same time the best method of confirming the diagnosis and of curing the supposed auditory impairment. Galvanic skin tests are also sometimes useful.

The Doerfler-Stewart test is a simple and effective method of identifying psychogenic deafness: The patient is tested audiometrically, in a sound-proof room, with words. As the test proceeds, noise of gradually increasing intensity is introduced into the room and careful note is made of when he can no longer hear the speaking voice. If his hearing is normal, or if his hearing defect is organic, he will be able to hear the words until the intensity of the noise either reaches the level of the speaking voice or exceeds it by 10 to 15 db. If he is malingering or is suffering from psychogenic deafness, he will stop hearing the speaking voice before the noise attains the same intensity.

According to Doerfler (9), the audiograms of psychogenic deafness are of the saucer type, usually lying between 50 and 90 db. The nature of the slope, which is inclined to rise at both ends, made him suspect that the individual utilizes, in effect, an equal loudness contour in maintaining the level of hearing which he demonstrates. This type of saucer audiogram was present in 80 per cent of Doerfler's psychogenically deaf patients.

SUMMARY

The full magnitude of the problem of deafness in the United States is unknown but there is no doubt that it has materially increased as more and more persons have lived into the upper age brackets. The attitude of otologists toward the subject of deafness has materially changed in recent years. Only a small number of totally deaf persons can be cured, and the hearing of only a limited number can be improved, but aural rehabilitation offers a new outlook for all. The practitioner of geriatrics can do much to teach these facts to patients and to guide them to otologic consultation. This requires a background knowledge of diagnostic and etiologic facts. The routine of diagnosis includes history taking, physical examination, local examination, tuning-fork tests and audiometric tests. After a specific case of deafness is classified as conductive, perceptive, or

mixed, etiologic factors are investigated. Presbycusis is the most frequent cause of deafness in older persons, but there are many other causes, fundamentally related to the patient's earlier life; these include otitis media, otosclerosis, syphilis, Ménière's disease, acoustic trauma, drugs, neoplasms, cerebral injury and vascular disease. Psychogenic deafness should always be the last explanation to be accepted.

REFERENCES

1. Beasley, W. C.: The general problem of deafness in the population, *Laryngoscope* **50:** 856 (Sept.) 1940.
2. McLaurin, J. W.: Otolaryngology today; its limitations and opportunities, *J. Arkansas M. Soc.* **50:** 98 (Nov.) 1953.
3. Morrissett, L. E.: Plight of the nerve-deaf patient; the uselessness of all present therapy; the practical usefulness of aural rehabilitation, *Arch. Otolaryng.* **51:** 1 (Jan.) 1950.
4. Leisti, T. J.: Audiometric studies of presbyacusis, *Acta oto-laryng.* **37:** 555, 1949.
5. Saxén, A.: Inner ear in presbyacusis, *Acta oto-laryng.* **41:** 213, 1952.
6. Fowler, E. P.: Emotional factors in otosclerosis, *Laryngoscope* **61:** 254 (Mar.) 1951.
7. Tamari, M. J., and Itkin, P.: Penicillin and syphilis of the ear, *Eye, Ear, Nose & Throat Month.* **30:** 252 (May) 1951; **30:** 301 (June) 1951; **30:** 358 (July) 1951.
8. Fowler, E. P.: Sudden deafness, *Ann. Otol., Rhin. & Laryng.* **59:** 980 (Dec.) 1950.
9. Doerfler, Leo G.: Psychogenic deafness and its detection, *Ann. Otol., Rhin. & Laryng.* **60:** 1045 (Dec.) 1951.

PROBLEMS ARISING IN THE MANAGEMENT OF SENILE CATARACT

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The purpose of this paper is to present some of the problems that arise when the ophthalmologist discovers that a patient has a senile cataract.

A cataract is an opacity of the crystalline lens. It has been estimated by various observers that there is some degree of opacity of the lens in 65 to 98 per cent of people past 60 years of age (1). Cataract is a term which the ophthalmologist uses very sparingly in talking to a patient, because opacities of the lens vary so greatly in extent. Some occupy the whole lens, whereas others are present in only a few spots; some are dense and definitely obstruct vision, whereas others are less dense and obstruct vision very little. If they are in the central part of the lens behind the pupil, they may obstruct vision severely; on the other hand, even when the portion of the lens behind the pupil is encroached upon, vision may be decreased only slightly because the rays of light are still able to pass through some clear lens and reach the retina. Most cataracts progress very slowly if they are not due to injury, to diabetes or to some other eye disease such as uveitis or tumor. The usual cataract not due to these causes, occurring in the aged, is called a senile cataract, and most of them do not cause sufficient visual disability before death to necessitate removal by surgery.

DOCTOR AND THE PATIENT

The slow growth of these senile cataracts and the age of the patient are the reasons for the ophthalmologist's reluctance to use the word 'cataract', unless necessary. It is not very sensible to tell a 70-year-old man with a life expectancy of about ten years that he has a cataract, when he has vision of 20/30 and some lens opacities that may not reduce vision sufficiently to need surgery for ten to twenty years. To most patients the word cataract means blindness and painful surgery. When it seems that surgery will not be necessary for a long time, the ophthalmologist need not inform the patient about the cataract unless he fears a complication such as glaucoma. When the doctor fears the onset of glaucoma, it may be necessary to mention the cataract in order to have a careless patient return for a routine check. Glaucoma may develop silently and painlessly, unnoticed by the patient until too late, unless discovered by the doctor at the routine visit. The ophthalmologist may suspect the development of glaucoma when the eye has a narrow angle or a shallow anterior chamber. If there is noticeable loss of vision but not enough to justify surgery, he will be called upon to make an explanation, which is often difficult without the use of the word cataract. The ophthalmologist usually tells the patient that there is some hardening of the lens or some spots in the eye. He may also tell the patient that it is part of the process of getting older and, just as one no longer walks as fast as when young,

neither does one see as sharply. It should be emphasized to the patient that the condition does not mean that there will be complete loss of vision. It is necessary to give assurance to older people with cataracts.

When the word cataract must be used, the patient should be told that of all the conditions that cause visual loss necessitating surgery, the one most amenable to treatment is cataract, because of its high percentage of surgical cure.

NONSURGICAL TREATMENT OF CATARACTS

When the patient is told that he has a cataract, he will usually ask if there is any other treatment besides surgery, because his friends have told him that drops keep a cataract from growing or that "injections of fish eyes" cure cataracts. He must be told that there is no cure except surgery. At the same time he should be examined by his family doctor for diabetes and also have a general physical examination. He should also be told that unproven treatments may be harmful. Injections of lens antigen from other animals may cause a sensitivity to the patient's own lens protein, because lens protein is an organ-specific allergen (2). Then, when the patient's cataract is removed, a uveitis may develop, with sequelae which may progress even to blindness (3).

WHEN TO OPERATE ON CATARACTS

A patient with opacities of more than the ordinary degree should be checked every six months. The decision about when to remove a cataract depends on many factors. For many years cataracts were not removed until they were mature or "ripe", because maturity facilitated the extracapsular method of extraction. However, this is no longer the criterion, as the intracapsular operation can be performed in older patients and the cataract does not have to be mature. In the case of the employed patient, the cataract is removed as soon as he is unable to carry on with his occupation; the eye with the worse vision is operated on first, if it has good potential vision. In the case of the unemployed but mentally alert patient, surgery is performed when vision in the better eye is reduced to 20/70. Some surgeons will operate on all cataracts when they mature, regardless of the amount of vision in the better eye, in order to give the patient side vision and to prevent hypermaturity of the cataract and the possibility of complications that accompany it. Most ophthalmologists prefer not to remove a cataract if the vision in the other eye is good, because after the cataract is removed on only one side, the eyes no longer work together as a unit. This is because, after the cataract is removed, a thick, high convex type of correcting glass is required in order for an eye to see as sharply as possible. This lens magnifies the objects at which this aphakic eye looks. However, to the eye which has not been operated on, the objects appear much smaller. The brain is unable to fuse a small image and a much larger image and the patient either sees double or sees with only one eye, suppressing the image in the other eye. Because of this, after a cataract has been removed, both eyes are provided with high convex lens. This lens blurs vision in the eye which has not been operated on, so that it is easier for the brain to suppress the image and thus help to prevent double vision.

One of the difficult decisions to make is when to remove a cataract from a patient's one useful eye. Operation is delayed as long as feasible because of the possibility of unsuccessful results, which might leave the patient with less vision than before the operation, even to the point of total blindness. Nevertheless, a little vision is better than no vision. In the case of employed people who must earn a livelihood, the operation is postponed until they are unable to see well enough to perform their tasks. In the case of older and unemployed patients, various criteria are used. Some surgeons operate when the patient cannot walk safely without help. Others wait until the patient is unable to recognize the faces of people he knows. The decision is frequently very difficult.

DISEASES CONTRAINDICATING CATARACT EXTRACTION

Before operating for senile cataract the question arises as to whether the patient is in good enough physical condition to withstand the extraction. There is practically no mortality from the operation itself, as it is usually performed under local anesthesia; there is little or no shock and the amount of blood lost is infinitesimal. The constitutional conditions that cause operative complications are cough, asthma, bronchitis, enlargement of prostate with dysuria, psychosis, diabetes with extremely high blood sugar levels, hemorrhagic diseases, and uncontrollable hypertension.

Cough, asthma, bronchitis, and dysuria make operation difficult because they cause an increase of intravenous pressure. This increase is conducive to expulsion of the intraocular contents during surgery. Uncontrolled diabetes and extreme hypertension are conducive to hemorrhage. Patients with psychoses cannot give the cooperation necessary after a cataract operation.

Old age is no contraindication to cataract extraction, if the patient has a reasonable life expectancy. Moderate hypertension is also no contraindication. Cardiac conditions are contraindications only if there is very marked dyspnea or cough, or if the life expectancy is very short.

There have been instances in which the family doctor has rendered a disservice by saying that the patient is too old to have his cataracts removed or that the patient's heart cannot stand an operation. Sometimes after a patient has heard this, he absolutely refuses an operation and goes through the rest of his life as a blind man, although really well enough to have had the cataract removed. Although the family doctor meant well, a word lightly spoken was sufficient to prevent this patient from ever seeing again. The doctor would have done much more for the patient if he had consulted an ophthalmologist first.

Patients do not die from cataract surgery under local anesthesia. Moreover, many postoperative systemic complications may be avoided, as nowadays patients may frequently be permitted out of bed the day after the operation, because of the use of corneoscleral sutures that produce a strong, tight wound closure.

POSTOPERATIVE DIFFICULTIES

After the operation, the patient still has many obstacles to overcome. Objects appear larger, and therefore closer than normal. The postoperative cataract

patient finds that he is clumsy with the most familiar tasks he must perform. If he does not look through the center of the glasses, objects are blurry. Lines which are straight appear curved. All these difficulties are gradually overcome. However, a portion of the surroundings is not seen, because of a scotoma or blind area between the central and peripheral visual fields. To add to the patient's discomfort, the glasses are very heavy. They constantly need adjustment and if they are not centered exactly, vision is distorted. Alas, aside from vision, the only compensation the postoperative cataract patient gets is that the sky looks very blue; sometimes they even complain of this. These facts plus the desire of the 75-year-old patient to see as well as when he was 20 years old, call into play not only the science, but the art of medicine to the nth degree.

REFERENCES

1. Duke-Elder, W. S.: Text-Book of Ophthalmology. Vol. III. Diseases of the Inner Eye. St. Louis, C. V. Mosby Co., 1941, p. 3193.
2. Duke-Elder, W. S.: Text Book of Ophthalmology. Vol. I. The Development, Form and Function of the Visual Apparatus. London, Kimpton; and St. Louis, C. V. Mosby Co., 1932, p. 485.
3. Report of the Committee on Ophthalmology of the Division of Medical Science, National Research Council, *J.A.M.A.* **148:** 707 (June 20) 1953; and Editorial, p. 710.

MANAGEMENT OF BENIGN HYPERTROPHY OF THE PROSTATE

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Hypertrophy of the prostate with all its sequelae is becoming of increasing importance as the number of individuals over the age of 50 increases. At the University Hospitals in Iowa City in the past twenty years, approximately 13,000 patients requiring therapy for this disease were seen. In the last five years there were more than 4,000 patients with symptoms of bladder-neck obstruction due to benign prostatic hypertrophy, so that the number of patients with this particular problem is increasing.

Although the exact figures on the incidence of prostatic hypertrophy are difficult to obtain, various studies have been made which give the following incidence (1):

Age	No. of prostates examined	Per cent with BPH	Per cent with BPH and Ca
21–30	24	0%	0%
31–40	28	4%	0%
41–50	23	30%	14%
51–60	65	37%	21%
61–70	77	67%	23%
71–80	63	68%	14%
81–90	24	75%	27%

Reproduced (modified) with permission, from Cowdry's Problems of Ageing edited by A. I. Lansing (1).

The problems which bring the patient to the physician and require therapy are 1) hematuria from the enlarged prostate, and 2) bladder-neck obstruction with its sequelae. In about 50 per cent of the patients, enlargement of the prostate due to benign hypertrophy requires no therapy, since hematuria or evidences of bladder-neck obstruction are absent.

PATHOLOGY

The pathologic process is essentially a nodular hyperplasia of the inner portion of the prostate. This compresses the outer portion, producing a narrow rim of tissue outside the hypertrophied area, which contains the uninvolved prostatic glands. At any time, this outer portion may become the seat of a carcinoma or of a new hypertrophy which will recreate the difficulties with which the patient presented himself. The inner hypertrophied portion causes rather pronounced distortion of the urethra and bladder neck, giving rise to obstruction and difficulties in urination. Large veins may usually be seen coursing along the urethral surface of the enlarged prostate, and these frequently are the source

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of gross hematuria. As more and more distortion occurs, obstruction to the outflow of urine becomes greater, and results in hypertrophy of the bladder musculature, trabeculation of the bladder wall, diverticulum formation, infection, stone formation, and finally damage to the upper urinary tract, as manifested by hydroureter, hydronephrosis, and severe renal insufficiency.

The extent to which these sequelae occur, or are present at the time the patient presents himself, will influence the prognosis greatly. The presence or absence of these manifestations of prostatic obstruction also modifies the symptomatology, makes diagnosis easy or difficult, and influences the type of therapy which is to be carried out. If the changes due to obstruction are very great the patient may have some persistent symptoms and signs, even though the hypertrophied tissue has been adequately removed. At times, the progress of these changes is so insidious that the patient will have few symptoms. It is, therefore, a good plan to have every man over the age of 60 undergo intravenous pyelography every four months, to study the character of the bladder and upper urinary tract. Many of these individuals will have benign enlargement of the prostate. Since many of them will have very few symptoms but will begin to show extensive changes in the bladder wall before infection and symptomatology occur, these changes should be sought for in the excretory cystogram.

SYMPTOMS AND SIGNS

The patient may present himself to the physician with any of the following manifestations: gross hematuria, frequency and difficulty of urination, evidences of urinary-tract infection, the presence of a suprapubic mass indicating a large amount of residual urine, or signs and symptoms of renal insufficiency. The latter may frequently be confused with symptoms of gastrointestinal disease, namely, loss of appetite, loss of weight and, in late cases, nausea and vomiting. In all such instances, the possibility of renal insufficiency should be considered, and appropriate renal function tests carried out. Intravenous pyelography, excretory and retrograde cystography and urethrography are all valuable aids in the evaluation of the degree of pathologic change. The presence or absence of residual urine can be determined by excretory cystography, if one does not wish to catheterize the patient. However, catheterization is usually desirable, and this can be carried out by having the patient void and then passing a small, # 16 French, Coudeé-tipped catheter, using plenty of lubricant in the urethra in order to avoid trauma. If the catheter does not pass easily into the bladder, excretory cystography should be carried out first.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis between bladder-neck obstruction due to benign hypertrophy of the prostate and neurogenic bladder must also be made, particularly in older individuals with a good deal of cerebral arteriosclerosis. In these men, so-called uninhibited neurogenic bladder will cause frequency of urination, but not retention of urine. Usually intravenous pyelography with a careful study of the excretory cystogram will differentiate obstructive uropathy from the un-

inhibited neurogenic bladder. The latter will show a perfectly smooth bladder wall, with none of the trabeculation, hypertrophy of the muscle fibers, and small-diverticulum formation seen so characteristically in the obstructive situation. The uninhibited neurogenic bladder will improve upon the administration of belladonna or one of the belladonna-like drugs. On the other hand, the administration of belladonna to a patient with beginning bladder-neck obstruction will bring about complete retention of urine. Rectal examination is extremely important in differentiating hypertrophy of the prostate from carcinoma of the prostate. If the latter is present, as manifested by the characteristic stony hardness, a different type of therapy is indicated.

TREATMENT

Having made a diagnosis of benign hypertrophy with obstruction to the outflow of urine, the following facts should be carefully ascertained: 1) The general condition of the patient, *e.g.*, his age, cardiovascular status, and fluid balance. 2) The facts with regard to the local situation. How big is the prostate and what type of distortion is present? Is the enlargement a large median lobe or two large lateral lobes; and is it mostly intraurethral or intravesicular? How much infection is there in the urethra, prostate, bladder, and upper urinary tract, and how much residual urine is present? What is the extent of trabeculation and diverticulation of the bladder? Is there an associated vesical calculus? How much damage has been done to the upper urinary tract? Is there ureteral reflux? An evaluation of the total situation will then determine therapy.

If there is marked infection, with long-standing urinary-tract obstruction resulting in upper urinary-tract damage and uremia, it is well to institute drainage by means of an indwelling urethral catheter or suprapubic cystostomy, meanwhile controlling the infection and improving renal function, before attempting to do anything about the prostate. If electrolyte and water balance are disturbed, they should be corrected. Hemorrhage, if present, necessitates immediate control. Sometimes this may be accomplished by an indwelling urethral catheter of the Foley type; at other times, however, immediate prostatectomy is indicated in order to control the hemorrhage.

Having improved the patient's general condition, having controlled infection during temporary indwelling catheter or suprapubic cystostomy drainage, consideration is then given to the removal of the enlarged portion of the prostate. Prostatectomy may be carried out by one of four techniques: suprapubic, transurethral, retropubic, or perineal. All of these operations are satisfactory for the removal of the hypertrophied prostate. All have their own indications and contraindications because of their respective difficulties, complications and late sequelae.

Perineal prostatectomy, for example, is followed by a greater incidence of incontinence, and there is a possibility of stricture at the bladder neck. However, it lends itself readily to the early diagnosis of a suspicious nodule in the prostate; if the nodule proves to be carcinoma, radical prostatectomy may be then carried out. The perineal operation is useful, therefore, as a procedure in an

individual who is over 60 and who has an enlarged prostate which is suspected to be carcinomatous. Retropubic prostatectomy and suprapubic prostatectomy are suitable for the very large benign enlargement, particularly when it projects into the bladder. The average moderately enlarged prostate, and often the very large one, is adequately and safely treated by transurethral prostatic resection. Particularly is this true if there are no significant vesical calculi, and no diverticula, or urethral or ureteral reflux. Especially with the smaller enlargements, even patients over 90 are readily managed by transurethral prostatectomy. In the presence of diverticula or fairly large stones, it is usually more satisfactory, except in very obese or very old individuals, to carry out a suprapubic prostatectomy and simultaneously take care of the associated condition. None of these operations is prophylactic for carcinoma of the prostate, as in 95 per cent of the cases the malignant neoplasm starts in the outer portion of the prostate. Total prostatectomy would be the only prophylactic type of prostatectomy for carcinoma of the prostate. It is generally felt that total prostatectomy should not be carried out for benign hypertrophy, because of the specific complications —primarily impotence, and an increased incidence of incontinence.

The mortality and morbidity rates for the surgical management of individuals with nodular hyperplasia of the prostate vary with the factors involved, as outlined, but in general they are surprisingly low. Large series have been reported with a mortality rate in the neighborhood of 1 per cent. If evidences of obstruction are recognized early before extensive sequelae of an irreparable nature occur, present-day methods of surgical management show a mortality rate of even less than 1 per cent.

Good nursing care is extremely important. Many aged patients who get along relatively well in the familiar atmosphere and routine of their homes become thoroughly confused and dependent in the strange, new atmosphere of the hospital. The nurse should aid them in their adaptation to the new environment and literally spoon-feed those who require it. The nurse, like the physician, should be aware of the other chronic diseases that her charges have, such as blindness, deafness, paralysis, diabetes, arteriosclerosis or cardiac disease, and should modify her nursing care to accommodate the particular needs of the individual patient. The diet should be adjusted to the patient's needs and condition. Catheters should be checked many times daily to be sure that they are draining. Many patients have had stormy postoperative courses because a plugged catheter was not noted until the patient began to bleed massively or had severe chills and fever. Accurate, reliable records of intake and output, temperature, blood pressure, and variations in the patient's normal behavior pattern, are imperative.

Before and immediately after leaving the hospital, a gradual increase in the patient's activity, with rehabilitation to the surroundings of his home, may be important. Since it takes about six weeks for repair of the prostatic urethra after any type of prostatectomy, there may be periods of infection and slight hematuria or an attack of epididymitis. This should be explained to the patient and to the individuals caring for him in this postoperative period.

SUMMARY

Hypertrophy of the prostate is a common lesion. It is increasing in frequency because of the increasing proportion of the population in the older age group. It originates in the inner portion of the prostate, compressing the normal prostate gland posteriorly, and giving rise to 1) hematuria, and 2) bladder-neck obstruction with its sequelae of stone formation, infection, renal insufficiency, and distortions of the anatomy of the bladder and upper urinary tract. Its management varies with the age and general condition of the patient and with the extent of changes which have been brought about by its presence. Treatment involves first attaining kidney and bladder drainage equilibrium, and then correcting infection, improving renal function and correcting, as much as possible, the sequelae of the obstruction which has been present. In addition, it involves removing the cause of the obstruction, the hypertrophied portion of the prostate. This can be accomplished by four types of surgical procedure, which have been described. Once prostatectomy has been carried out, the patient should be followed regularly every four months, primarily by means of urine examinations and intravenous pyelograms, in order to be sure that no stricture formation, either in the prostatic or in the penile urethra, is producing further obstruction; and that other sequelae, if present, continue to improve. Nursing and rehabilitation problems are of extreme importance in many of these patients, primarily because of their age.

REFERENCE

1. LANSING, ALBERT I., Editor: Cowdry's Problems of Ageing, ed. 3. Baltimore, The Williams & Wilkins Company, 1952, p. 695.

THIRD INTERNATIONAL GERONTOLOGICAL CONGRESS—LONDON

The Third International Gerontological Congress will be held in London, England, *July 19–23, 1954*, under the presidency of Dr. J. H. Sheldon. Arrangements by the British Organizing Committee are well advanced. Dr. R. E. Tunbridge, B.M.A. House, Tavistock Square, London W.C.1, is Chairman of the Committee. An *American Committee* is cooperating with the British Committee, especially in regard to the program. Of this Committee *Dr. W. B. Kountz, 660 S. Kingshighway Blvd., St. Louis 10, Mo., is the Chairman.* The Congress will be both instructive and enjoyable. It is imperative for those planning to attend and to present their work, to send titles of papers, together with abstracts of not more than 250 words, to Dr. Kountz. As in the case of other International Congresses, personal invitations to present papers are not necessary but in the event that these would be helpful in obtaining subsidies for travel, the American Committee will be happy to request the British Organizing Committee to send official invitations.

The Research Committee on Biology and Medicine will meet at the same time as the Third International Gerontological Congress. The Chairman of this Committee is Dr. W. O. Thompson, Chicago, Illinois.

Because the time is short and it is difficult to get transatlantic reservations, except the more expensive ones, blocks of reservations are being held by Convoys Travel Service, 1133 Broadway, New York 10, N. Y. All correspondence concerning *travel information* should be addressed to *Mrs. V. Kelly, 660 S. Kingshighway, St. Louis 10, Mo.*

PROVISIONAL PROGRAM

SATURDAY, JULY 17

6 to 8 P.M. Reception at the Apothecaries Hall for Overseas Members to meet Officials and Members of the Committees.

8 P.M. Dinner of the Governing Body at Apothecaries Hall.

SUNDAY, JULY 18

RELIGIOUS SERVICES

Members will be welcomed at Matins in Westminster Abbey, at the 10.30 Mass in Westminster Cathedral, and at Morning Services in Central Hall, Westminster (Methodist), and in St. Columba's, Pont Street (Presbyterian).

Any members wishing to attend a Service in the morning are requested to inform the British Organising Secretary before July 1.

AFTERNOON

Arrangements will be made for informal visits and hospitality in London and its environment for any member or delegate from overseas.

MONDAY, JULY 19

9 **A.M.** — onwards — Registration will take place in the Bishop Partridge Hall, Church House.

11 **A.M.** Assembly Hall—An introductory talk will be given to explain the nature of the technical visits taking place on Wednesday afternoon, July 21, and Saturday, July 24.

2 **to 6 P.M.** Opening Ceremony.

6 **to 7.30 P.M.** Reception by kind permission of Her Majesty's Government at Lancaster House for Overseas Members.

TUESDAY, JULY 20

9.30 to 11 A.M., 11.15 A.M. to 12.45 P.M., 2.30 to 4 P.M. Meetings of all Sections.
4.30 to 6.30 P.M. Plenary Session on "Critique of Surveys."
EVENING

Reception by kind permission of King Edward's Hospital Fund for London (limited numbers).

WEDNESDAY, JULY 21

9.30 to 11 A.M., 11.15 A.M. to 12.30 P.M. Meetings of Sections.
1.45 P.M. Coaches leave for special conference visits to hospital centres, including geriatric units, hostels and social work in relation to the aged.

THURSDAY, JULY 22

9.30 to 11 A.M., 11.15 A.M. to 12.45 P.M., 2.30 to 4 P.M. Meetings of Sections.
4.30 to 6.30 P.M. Plenary Session on "Problems of Employment of the Elderly."
8 P.M. Ballet Performance and Buffet Supper at the Royal Festival Hall for all Full and Associate Members.

FRIDAY, JULY 23

9.30 to 11 A.M., 11.15 A.M. to 12.45 P.M. Meetings of Sections.
12.30 P.M. Meeting of the Governing Body.
2.30 P.M. Closing Session of the Congress.
Prayer.
4 to 5.30 P.M. Garden Party by kind permission of the Nuffield Foundation at Nuffield Lodge.
6 to 8 P.M. Reception by kind invitation of the Rt. Hon. Lord Amulree at the House of Lords.

SATURDAY, JULY 24 and SUNDAY, JULY 25

Informal visits will be arranged for any member wishing to see Hospitals, Hostels, Housing, Accommodation, Clubs or any sociological activity in respect of the aged in the London area.

MONDAY, JULY 26

Commencement of official visits to Provincial Centres.

OFFICIAL VISITS

To selected places in the London area on Wednesday, July 21.

GENERAL VISITS

Every facility will be given to members to arrange for visits to centres of interest in the United Kingdom in relation to work on behalf of the aged, whether by Voluntary Societies, in Hospitals or under the care of Local Authorities.

TOURS

The technical/sightseeing Tours have been designed primarily to give visitors an opportunity to see the varied work being done for the elderly in hospitals, special housing sites, institutions, clubs and research centres. These tours will be personally conducted. A member of the Congress will accompany the party at all stages, and members will be officially received at each centre. In addition to seeing examples of every aspect of work on behalf of the elderly, places of historic interest and natural beauty will be visited.

PRE-CONGRESS SCOTTISH RAIL AND COACH TOUR-A— JULY 12 TO JULY 17 INCLUSIVE

Pitlochry is the base of the Tour, where delegates will stay four days visiting Dundee, Blair Atholl Castle, Glamis Castle, Loch Lomond, Stirling Castle, Edinburgh Castle and Holyrood Palace and Glasgow.

CONGRESS LONDON AND DISTRICT COACH TOUR–B— JULY 19 TO JULY 23 INCLUSIVE

Sightseeing Tours of London, Windsor Castle, Hampton Court Palace, Hatfield House, Hertfordshire.

For visitors who are taking no official part in the Congress

POST-CONGRESS ENGLISH COACH TOUR–C— JULY 26 TO AUGUST 3 INCLUSIVE

Visiting Oxford (night stop), Blenheim Palace, Stratford-on-Avon (stay two nights), Cheltenham Spa, Bristol (night stop), Cheddar, Wells, Taunton, Torquay (stay three for nights), Truro, St. Austell, Southampton (night stop), Winchester, Portsmouth, London.

POST-CONGRESS ENGLISH COACH TOUR–D— JULY 26 TO AUGUST 3 INCLUSIVE

Visiting Oxford, Blenheim Palace, Stratford-on-Avon (stay for two nights), Nottingham (night stop), Harrogate (stay for four nights), Yorkshire, Durham, Sunderland, York, Lincoln, Cambridge (night stop), London.

For further particulars of these Tours apply to the officially appointed travel agents:

CONVOYS LTD.

30 Bouverie Street, London, E.C.4

Telephone CENtral 7511

and at:

1133 Broadway, New York 10, N. Y., U. S. A.

Telephone WATKINS 9-0920

Early Reservations are Essential as Bookings for Tours will be Closed on April 1

MEMBERSHIP OF THE CONGRESS

FULL MEMBERSHIP permits a Member to attend all Sessions and all official functions of the Congress, including the Ballet and Buffet Supper on Thursday, July 22. It also includes the receipt of all official literature relating to the Congress except any post-conference report which may be published which would be available to Full Members at a special price.

SECTIONAL MEMBERSHIP permits the Sectional Member to attend any Session of the Congress, including official lectures and visits arranged in connection with the Congress, and to receive all official publications to the Congress except any report which may be printed after the Conference.

ASSOCIATE MEMBERSHIP is for the wife or husband of a Full Member and permits an Associate Member to attend all official visits and social functions of the Congress, including the Festival Ballet and Buffet Supper and the Opening and Closing Ceremonies of the Congress.

DAY MEMBERSHIP. Any individual wishing to attend the Sessions on one day only of the Conference may make special application and the fee per day will be £1.

NOTE. *These membership fees apply only to persons who apply before April* 1, *enclosing fees. The fees for application received after this date will be* £10, £6 £4, *and* £1 10*s. Certain of the Social functions may have to be limited in number. Where limitation is necessary, preference will be given to members who apply early.*

IF NOT ALREADY REGISTERED, PLEASE COMPLETE THE ENCLOSED APPLICATION FORM.

TUESDAY, JULY 20

TIME	SECTION 1 BIOLOGY AND PATHOLOGY Chairman: Sir Francis Frazer Secretary: Dr. O. Olbrich	SECTION 2 GERIATRIC MEDICINE AND PSYCHIATRY Chairman: Dr. Marjory Warren Secretary: Dr. Thomas Wilson	SECTION 3 SOCIOLOGY AND PSYCHOLOGY Chairman: Miss E. I. Black Secretary: Miss M. Bucke	SECTION 4 RESEARCH COMMITTEE Secretary: Dr. V. Korenchevsky
9.30 a.m. to 11 a.m.	SYMPOSIUM Nutrition-experimental aspects in relation to ageing.	Opening Session	Introduction to Social Policy Problems. (a) Demographic approach.	Round table discussion of schemes and methods for research on "Changes in cells with ageing, with a particular examination of the compensatory hypertrophy in the cells of tissues and organs in cases of degenerative changes and diseases accelerating the process of ageing." British Chairman: Professor J. E. Danielli. A full day session will be devoted to this.
11 a.m. to 11.15 a.m.	INTERVAL			
11.15 a.m. to 12.45 p.m.	Miscellaneous pathological communications.	SYMPOSIUM Nutrition—human aspects in relation to ageing.	(b) Translation of policy into practice. (c) Human aspects.	
12.45 p.m. to 2.30 p.m.	LUNCHEON INTERVAL			
2.30 p.m. to 4 p.m.	Problems of ageing in connective tissue.	Miscellaneous papers on disorders of the cardiac vascular system in the aged.	Comprehensive care	
4.30 p.m. to 6.30 p.m.	PLENARY SESSION—CRITIQUE OF SURVEYS			

WEDNESDAY, JULY 21

9.30 a.m. to 11 a.m.	SYMPOSIUM Endocrinological aspects of ageing with special reference to the female generative organs.	SYMPOSIUM Surgery of the elderly.	General Provisions A critical survey from various areas.	Round table discussion of schemes and methods proposed for research on: "Causes of mental inefficiency in the elderly." British Chairman: Dr. Macdonald Critchley. A full day session will be devoted to this.
11 a.m. to 11.15 a.m.	INTERVAL			
11.15 a.m. to 12.30 p.m.	(continued)	(continued)	(continued)	
12.30 p.m. to 1.45 p.m.	LUNCHEON INTERVAL			
1.45 p.m. to 4 p.m.	LONDON VISITS			

THURSDAY, JULY 22

9.30 a.m. to 11 a.m.	SYMPOSIUM *Part I.* The Experimental study of skill in the elderly.	Neurology in relation to old age with special reference to incontinence.	Psychology Various papers relating to psychological aspects of ageing.	Round table discussion of schemes and methods proposed for research on: "Genetical aspects of ageing."
11 a.m. to 11.15 a.m.	INTERVAL			
11.15 a.m. to 12.45 p.m.	Pathology of the cardio-vascular system in the aged.	SYMPOSIUM with Section III. Effect of ageing on mental and physical capacity.	SYMPOSIUM with Section II. Effect of ageing on mental and physical capacity.	British Chairman: Dr. E. B. Ford, F.R.S. (continued
12.45 p.m. to 2.30 p.m.	LUNCHEON INTERVAL			
2.30 p.m. to 4 p.m.	SYMPOSIUM *Part II.* Pathology. Effect of ageing on the nervous system.	Diseases of bones and joints.	Welfare aspects of disability in the aged.	(continued)
4.30 p.m. to 6.30 p.m.	PLENARY SESSION—EMPLOYMENT OF THE ELDERLY			

FRIDAY, JULY 23

9.30 a.m. to 11 a.m.	SYMPOSIUM Changes in total metabolism and in the oxygen supply of individual organs as affected by age.	Genetics, Haematology	General welfare problems	Round table conference on the ways and means for achieving the rapid and efficient progress in gerontological work.
11 a.m. to 11.15 a.m.	INTERVAL			
11.15 a.m. 2.30 p.m. to 3.30 p.m.	Closing Session of Section	Closing Session of Section	Closing Session of Section	

Concluding Session of the Third International Congress

Please Note: All papers to be given in French or English and the synopses to be sent in by April 1, 1954.

RESEARCH COMMITTEE ON BIOLOGY AND MEDICINE of the INTERNATIONAL ASSOCIATION OF GERONTOLOGY

REPORT OF MEETING OF THE AMERICAN DIVISION, SAN FRANCISCO, AUGUST 25, 1953

A short meeting of the American section of the Research Committee on Biology and Medicine of the International Association of Gerontology was held during the meeting of the Gerontological Society, Incorporated, in San Francisco at the Marks Hopkins Hotel, Tuesday, August 25, 1953. The following members were present:

Dr. Warren Andrew
Dr. E. V. Cowdry
Dr. John Esben Kirk
Dr. Albert I. Lansing
Dr. N. W. Shock
Dr. W. O. Thompson, Chairman

These members of the Committee expressed interest in the ideas already set forth by Dr. V. Korenchevsky concerning the objectives of the Research Committee. Further consideration of the problem at the time of the meeting of the International Congress in London, England, July 19–23, 1954, was considered desirable.

It was felt that elaborate plans for the meeting in London could not be made at the present time because no funds are available for traveling expenses of members of the Committee. In most instances, American investigators and clinicians who wish to attend the meeting in London must do so at their own expense.

W. O. THOMPSON, M.D.
Chairman, Research Committee on Biology and Medicine

AMERICAN GERIATRICS SOCIETY

THE 1954 ANNUAL MEETING

The 11th Annual Meeting of the American Geriatrics Society will be held at the Fairmont Hotel in San Francisco, June 17 through 19.

Hotel reservations should be made through the San Francisco Convention and Visitors Bureau, 200 Civic Auditorium, San Francisco 2, California. Members should reserve accommodations immediately, stating times of arrival and departure, because the hotels expect to be filled to capacity.

The Annual Business Meeting will be held in the San Francisco Room of the Fairmont Hotel, Thursday morning, June 17, at 9 o'clock. All scientific sessions will be held in the Terrace Room. The Annual Dinner is scheduled for the Nob Hill Room on Friday evening, June 18, at 8 o'clock, preceded by cocktails in the Cirque Room Lounge at 7 o'clock.

The program will cover many aspects of geriatric medicine. There will be several panel discussions on subjects of current interest.

Dr. Laurance W. Kinsell, Highland Alameda County Hospital, 2701 Fourteenth Avenue, Oakland 6, California, is in charge of local arrangements for the meeting.

W. O. Thompson, M.D.
President

SCIENTIFIC PROGRAM

FAIRMONT HOTEL

THURSDAY, JUNE 17, 1954

8:00 A.M. Registration (*Terrace Room Foyer*)
9:00 A.M. Annual Business Meeting (*San Francisco Room*)
2:00 P.M. General Session (*Terrace Room*)

Norris J. Heckel, M.D., *Presiding*

1. Significance and Management of Genital Prolapse in the Aged
   Goodrich C. Schauffler, M.D., Portland, Ore.
2. Uterine Bleeding After the Menopause
   C. L. Buxton, M.D., New Haven, Conn.
3. The Use of Estrogens After the Menopause
   E. Kost Shelton, M.D., Los Angeles, Calif.
4. Cancer of the Breast—A Curable Disease
   H. Glenn Bell, M.D., San Francisco, Calif.

Edward Henderson, M.D., *Presiding*

5. The Psychology of Retirement
   Edward B. Allen, M.D. and Hollis E. Clow, M.D., White Plains, N. Y.
6. Criteria for the Feasibility of Rehabilitation in the Geriatric Patient
   Edward J. Lorenze, M.D., White Plains, N. Y.

Panel Discussion

7. RECENT DEVELOPMENTS IN CARDIOLOGY
   George C. Griffith, M.D., Los Angeles, Calif., Moderator
   Francis L. Chamberlain, M.D., San Francisco, Calif.
   William Paul Thompson, M.D., Los Angeles, Calif.

FRIDAY, JUNE 18, 1954

8:00 A.M. Registration (*Terrace Room Foyer*)
9:00 A.M. General Session (*Terrace Room*)

Edward B. Allen, M.D., *Presiding*

8. The Influence of Surgery in the Aged on Surgery in the Young
   Robert Elman, M.D., St. Louis, Mo.
9. The Diagnosis and Treatment of Diseases of the Bladder in the Aged
   Norris J. Heckel, M.D., Chicago, Ill.
10. The Fate of the Cholescystostomy Patient
    Daniel M. Hays, M.D. and Frank Glenn, M.D., New York, N. Y.
11. The Medical and Surgical Management of Acute Diverticulitis in Geriatric Patients
    Robert Priest, M.D., Brock E. Brush, M.D. and Louis Gregory, M.D., Detroit, Mich.

Malford W. Thewlis, M.D., *Presiding*

12. New Trends in the Treatment of Fractures of the Hip in the Aged
    Ralph Soto-Hall, M.D., San Francisco, Calif.
13. Management of Hip Fracture in the Geriatric Patient
    Charles V. Heck, M.D., Chicago, Ill.

Panel Discussion

14. METHODS OF DETERMINING OPERABILITY IN OLDER PATIENTS
    Warren H. Cole, M.D., Chicago, Ill., Moderator
    Differences in Physiology of the Young and Aged: Their Application to Operability—*Arthur J. Hunnicutt, M.D., Oakland, Calif.*
    Cardiac and Renal Reserve in the Aged as Related to Operability—*Charles T. Stone, M.D., Galveston, Texas*
    The Use of Laboratory Tests in Determining Operability—*Dr. Cole*

INTERMISSION FOR LUNCH

2:00 P.M. General Session (*Terrace Room*)

Elizabeth Mason-Hohl, M.D., *Presiding*

15. Overtreatment in the Aged
    Malford W. Thewlis, M.D., Wakefield, R. I.

16. Clinical Experience in Restoring Verbal Communication to 266 Laryngectomized Patients by Esophageal Speech
Lynwood Heaver, M.D., Willard White and Nona Goldstein, B.A., New York, N. Y.
17. The Influence of Endocrine Substances on the Metabolism in the Older Individual
William B. Kountz, M.D. and Teofil Kheim, M.D., St. Louis, Mo.
18. A Study of the Effects of Thyroid Medication on Pathology and Longevity in Aged Human Beings
Paul Starr, M.D., Los Angeles, Calif.

Warren H. Cole, M.D., *Presiding*

19. Hormones, Growth and Senescence
Laurance W. Kinsell, M.D., Oakland, Calif.

Panel Discussion

20. CHANGES IN STEROID EXCRETION WITH ADVANCING YEARS
Cornelius P. Rhoads, M.D., New York, N. Y., Moderator
Technical Advances in Steroid Measurement—*Thomas F. Gallagher, Ph.D., New York, N. Y.*
The Chromatograms of Urinary 17-Ketosteroids in Aged Persons Before and After the Administration of Stanolone and Testosterone —*Thomas H. McGavack, M.D., Sidney Pearson, Ph.D. and Helmut Haar, M.D., New York, N. Y.*
Adrenocortical Capacity and Metabolism of Hydrocortisone in Elderly Patients—*Frank Tyler, M.D., Salt Lake City, Utah*

ANNUAL DINNER, Friday, June 18, 1954

7:00 P.M. Cocktails—Cirque Room Lounge—Sponsored by the Schering Corporation
(Attendance limited to holders of dinner tickets)

8:00 P.M. Dinner (Dress optional)— Nob Hill Room
The dinner wine will be supplied by Wente Wineries, and the dessert wine by Cresta Blanca Wineries.

SATURDAY, JUNE 19, 1954

8:00 A.M. Registration (*Terrace Room Foyer*)
9:00 A.M. General Session (*Terrace Room*)

Hollis E. Clow, M.D., *Presiding*

21. Management of the Burned Elderly Patient
Sidney E. Ziffren, M.D., Iowa City, Iowa
22. Histologic Patterns of the Gastrointestinal Tract in Various Age Groups
Edward Henderson, M.D., Bloomfield, N. J.
23. A New Therapeutic Approach to the Management of Debilitating Diseases
Cortez F. Enloe, Jr., M.D., New York, N. Y.

Francis M. Pottenger, Jr., M.D., *Presiding*

Panel Discussion

24. THE RELATION OF INDUSTRY TO GERIATRICS
Carl M. Peterson, M.D., Chicago, Ill., Moderator

WHO SHOULD RETIRE?—*Charles E. Dutchess, M.D., New York, N. Y.*
THE IMPORTANCE OF GERIATRICS IN INDUSTRIAL MEDICINE—*Rutherford T. Johnstone, M.D., Los Angeles, Calif.*
THE STRESS DISORDERS—*William P. Shepard, M.D., New York, N. Y.*

25. TEMPORAL ARTERITIS
Bayard T. Horton, M.D. and Thomas B. Magath, M.D., Rochester, Minn.
26. PROGNOSIS AND TREATMENT OF OCULAR DEFECTS OF THE AGED
A. E. Maumenee, M.D., San Francisco, Calif.

TO BE READ BY TITLE

27. TUBE FEEDING WITH LIQUEFIED WHOLE FOOD IN ELDERLY PATIENTS
J. Barron, M.D. and L. S. Fallis, M.D., Detroit, Mich.
28. USE OF PSYCHOLOGIC TESTS IN DETERMINING PROGNOSIS AND TREATMENT IN GERIATRIC MENTAL ILLNESSES
D. V. Conwell, M.D., C. J. Kurth, M.D. and P. G. Murphy, Ph.D., Wichita, Kans.
29. MARRIAGE COUNSELLING IN GERIATRICS
A. K. Daniels, M.D., San Francisco, Calif.
30. STUDIES OF PLASMA HEPARIN LEVELS IN RELATION TO SEX, AGE, AND SERUM LIPIDS AND LIPOPROTEINS
H. Engelberg, M.D., Los Angeles, Calif.
31. BACTERIAL ALLERGY AS A CAUSE OF SO-CALLED INTRINSIC ASTHMA IN THE ELDERLY PATIENT
J. Forman, M.D. and H. Blatt, M.D., Columbus, Ohio
32. ARTERIAL DISEASE IN THE AGED
R. S. Gilfillan, M.D., San Francisco, Calif.
33. X-RAY FINDINGS IN GERIATRICS SURVEYS
G. J. Heppner, M.D., San Francisco, Calif.
34. STUDY OF THE PROBLEM OF PROSTHETIC FITTING AND TRAINING OF THE GERIATRIC PATIENT
M. K. Newman, M.D., Detroit, Mich.
35. SCREENING IN GERIATRICS
R. M. Perlman, M.D., San Francisco, Calif.
36. GERIATRIST-PHYSIATRIST TEAM IN GERIATRICS REHABILITATION
R. M. Perlman, M.D. and S. M. Dorinson, M.D., San Francisco, Calif.
37. BALLISTOCARDIOGRAPHY IN GERIATRICS
B. Schneider, M.D., Danville, Pa.
38. FURTHER CLINICAL STUDIES OF THE EFFECTS OF METRAZOL AND NICOTINAMIDE ON PSYCHIC AND MENTAL DISORDERS OF THE GERIATRIC PATIENT
Herman Seidel, M.D., Abraham A. Silver, M.D., Henry Nagel, M.D. and Jerome J. Blumberg, M.D., Baltimore, Md.

SCIENTIFIC SESSIONS ARE OPEN TO THE MEDICAL PROFESSION AND TO THOSE WHO ARE ESPECIALLY INTERESTED IN THE PROBLEMS OF AGING

GERONTOLOGICAL SOCIETY, INC.

The 1954 Annual Meeting of the Gerontological Society, previously scheduled for September 10–12, 1954, has been changed to December 28–30, 1954. The place of the meeting, Gainesville, Florida, remains unchanged.

N. W. Shock, Ph.D.
Secretary

WINNERS OF 1953 SCHERING AWARD FOR MEDICAL STUDENTS

Medical authorities in their special fields have selected the three winning papers in the 1953 Schering Award competition for medical students, according to an announcement by Dr. George Babcock, chairman of the Award committee. A $500 prize goes to the winner for each of three topics.

Two of the winners expressed professional interest in research. *George Richard Pieper*, junior at the University of Utah College of Medicine, wrote on "Antihistaminic Treatment of Upper Respiratory Allergies and Infections." He lives in Salt Lake City with his wife, who is a nurse, and their child. He did his premedical work at the Universities of Illinois and Missouri. After graduation, he plans internship first and later academic medical research in pharmacology.

Frank Edward Young, sophomore at the State University of New York at Syracuse College of Medicine, wrote on "Hormone Therapy of the Degenerative Diseases." His home is in Merrick, Long Island. His premedical training was at Union College in Schenectady. He hopes to interne at Presbyterian Medical Center but has not decided whether to go into general practice or research.

Irvin Paul Pollack won the $500 first prize for "New Concepts in the Treatment of Peptic Ulcer." He is a sophomore at the University of Maryland College of Medicine. He did his premedical work at Johns Hopkins, has a particular interest in genetics, and plans to interne. His home is in Baltimore, and he is an accomplished pianist.

Dr. Babcock said that of the three topics, "New Concepts in the Treatment of Peptic Ulcer" proved the most popular among last year's student authors, with 62 per cent writing on peptic ulcer, 21 per cent on degenerative diseases and 17 per cent on upper respiratory allergies.

BOOK REVIEW

Our Advancing Years. By TREVOR H. HOWELL, M.D. New York, N. Y., The Macmillan Co., 1954. $3.50.

Trevor Howell always has something worth while to write about. For years he has specialized in geriatrics, in hospitals. He is now working on combined social and medical rehabilitation of frail old people.

This book of 192 pages, well illustrated, is for the layman. Physicians will find it valuable too.

There are ten chapters including population trends, normal old age, old folks at home, organizations, hostels and institutions, aged sick, the story of geriatrics, geriatric organization, conclusions and recommendations.

Dr. Howell's expert handling of older people is interesting. He finds a lack of response to logical reasoning in the aged. It is a waste of time to appeal to reason. Rational trains of thought are almost impossible because the mental processes of advancing years are influenced by past experience, by suspicion, by previous modes of thought and by subconscious dread of the future. The author notes that old people can be persuaded to act on emotional grounds; they can be swayed by selfish motives. "They will sometimes obey a direct order or command; but listen to reason—never." The aged have lost their power of self-criticism. The author finds that old people are suspicious. This being a natural outcome of senescence explains why some old people have paranoid tendencies. Not all old people are like this, of course. Some are quite youthful in their approach to old age.

Dr. Howell's approach to the problems of old age is stimulating. His previous medical books are valuable; now he shows the practical problems that one faces in dealing with the aged at home or in institutions. His advice is that old people be gainfully occupied.

M. W. T.